

Current Clinical Strategies

Surgery

The UCI Manual of Surgery

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Surgical Documentation

Surgical History

Identifying Data: Patient's name, age, race, sex; referring physician; hospital identification number.

Chief Complaint: Reason given by patient for seeking surgical care; place patient's complaint in "quotation marks."

History of Present Illness (HPI): Describe the course of the patient's illness, including when it began, character of the symptoms; pain onset (gradual or rapid), precise character of pain (constant, intermittent, cramping, stabbing, radiating); other factors associated with pain (defecation, urination, eating, strenuous activities); location where the symptoms began; aggravating or relieving factors. Vomiting (color, content, blood, coffee-ground emesis, frequency, associated pain). Change in bowel habits; bleeding, character of blood (clots, bright or dark red), trauma; recent weight loss or anorexia; other related diseases; past diagnostic testing.

Past Medical History (PMH): Past medical problems. Previous operations and indications; dates and types of procedures; serious injuries, hospitalizations; diabetes, hypertension, peptic ulcer disease, asthma, heart disease; hernia, gallstones.

Medications: Aspirin, anticoagulants, hypertensive and cardiac medications, diuretics, anticonvulsants.

Allergies: Penicillin, codeine, iodine, others.

Family History: Medical problems in relatives. Family history of colonic polyposis, carcinomas.

Social History: Alcohol, smoking, drug usage, occupation.

Review of Systems (ROS):

General: Weight gain or loss; loss of appetite, fever, fatigue, night sweats. Activity level.

HEENT: Headaches, seizures, sore throat, masses, dentures.

Respiratory: Cough, sputum, hemoptysis, dyspnea on exertion, ability to walk up flight of stairs.

Cardiovascular: Chest pain, orthopnea, claudication, extremity edema.

Gastrointestinal: Dysphagia, vomiting, abdominal pain, hematemesis, melena (black tarry stools), hematochezia (bright red blood per rectum), constipation, change in bowel habits; hernia, hemorrhoids, gallstones.

Genitourinary: Dysuria, hesitancy, hematuria, discharge; impotence, prostate problems.

Gynecological: Last menstrual period, gravida, para, abortions, length of regular cycle birth control.

Skin: Easy bruising, bleeding tendencies.

Neurological: Stroke, transient ischemic attacks, weakness.

Surgical Physical Examination

Vital Signs: Temperature, respirations, heart rate, blood pressure, weight.

Eyes: Pupils equally round and react to light and accommodation (PERRLA); extraocular movements intact (EOMI).

Neck: Jugular venous distention (JVD), thyromegaly, masses, bruits;

lymphadenopathy; trachea midline.

Chest: Equal expansion, dullness to percussion; rales, rhonchi, breath sounds.

Heart: Regular rate and rhythm (RRR), first and second heart sounds; murmurs (grade 1-6), pulses (graded 0-2+).

Breast: Skin retractions, tenderness, masses (mobile, fixed) nipple discharge, erythema, axillary or supraclavicular node enlargement.

Abdomen: Contour (flat, scaphoid, obese, distended); scars, bowel sounds, bruits, tenderness, masses, liver span; splenomegaly, guarding, rebound, percussion note (tympanic), pulsatile masses, costovertebral angle tenderness (CVAT), abdominal hernias.

Genitourinary: Inguinal hernias, testicles, varicoceles; urethral discharge.

Extremities: Edema (grade 1-4+); cyanosis, clubbing, pulses (radial, ulnar, femoral, popliteal, posterior tibial, dorsalis pedis; simultaneous palpation of radial and femoral pulses). Grading of pulses: 0 = absent; 1+ weak; 2+ normal; 3+ very strong.

Rectal Exam: Masses, tenderness hemorrhoids, guaiac test for occult blood; prostate masses; bimanual palpation.

Neurological: Mental status; gait, strength (graded 0-5); tendon reflexes, sensory testing.

Laboratory Evaluation: Electrolytes (sodium, potassium, bicarbonate, chloride, BUN, creatinine), blood glucose, liver function tests, PT/PTT, CBC with differential; X-rays, ECG (if older than 35 yrs or cardiovascular disease), urine analysis.

Assessment (Impression): Assign a number to each problem and discuss each problem separately. Begin with most important problem and rank in order.

Plan: Describe surgical plans for each numbered problem, including preoperative testing, laboratory studies, medications, antibiotics, preoperative endoscopy.

Preoperative Preparation of the Surgical Patient

1. Review patient's history and physical examination, and write a preoperative note assessing patient's overall condition and operative risk.
2. **Preoperative Laboratory Evaluation:** Electrolytes, BUN, creatinine, PT/PTT, CBC, platelet count, UA, ABG, pulmonary function test. Chest X-ray (if patient has not had a normal chest x-ray in past 6 months or if >35 yrs old), EKG, (if older then 35 yrs old or if cardiovascular disease). Type and screen for an appropriate number of units of blood.
3. **Skin Preparation:** Patient to shower, and scrub the operative site with germicidal soap (Hibiclens) on the night before surgery. On the day of surgery, hair should be removed with an electric clipper or shaved just prior to operation.
4. **Prophylactic Antibiotics or Endocarditis Prophylaxis If Indicated.**
5. **Preoperative Incentive Spirometry** on the evening prior to surgery may be indicated for patients with pulmonary disease.
6. **Thromboembolic** prophylaxis should be provided for high-risk patients.
7. **Diet:** NPO after midnight.
8. **IV and Monitoring Lines:** At least one 18-gauge IV for initiation of anesthesia. Arterial catheter and pulmonary artery catheters (Swan-Ganz)

if indicated. Patient to void on call to operating room.

9. Medications

Preoperative sedation as ordered by anesthesiologist. Maintenance medications to be given the morning of surgery with a sip of water. Diabetics should receive one half of their usual AM insulin dose, and a insulin drip should be initiated with hourly glucose monitoring.

10. Bowel Preparation

Bowel preparation required for upper or lower GI tract procedures.

Antibiotic Preparation for Colonic Surgery

Mechanical Prep: Day 1: Clear liquid diet, laxative (milk of magnesia 30 cc or magnesium citrate 250 cc), tap water or Fleet enemas until clear.

Day 2: Clear liquid diet, IV fluids, NPO, laxative. Day 3: Operation.

Whole Gut Lavage: Polyethylene glycol electrolyte solution (GoLyte).

Day 1: 2 liters PO or per nasogastric tube over 5 hours. Consider adding metoclopramide (Reglan) 10 mg PO prn nausea; clear liquid diet.

Day 2: Operation.

Oral Antibiotic Prep: One day prior to surgery, after mechanical or whole gut lavage, give neomycin 1 gm and erythromycin 1 gm PO at 1 p.m., 2 p.m., 11 p.m. May substitute metronidazole 500 mg PO for erythromycin.

11. Preoperative IV Antibiotics: Initiated immediately preoperatively; consider additional dose during operation and 1 dose of antibiotic postoperatively to 24 hours post-op. Cefotetan (every 12 hours) for bowel flora, or first generation cephalosporin (Cefazolin) for clean procedures.

12. Anticoagulants: Discontinue Coumadin; stop IV heparin 6 hours prior to surgery.

13. Consider Stress Dose Steroids: Hydrocortisone 100 mg at midnight and at induction if history of steroid medication.

Admitting and Preoperative Orders

Admit to: Ward, ICU, or preoperative room.

Diagnosis: Intended operation and indication.

Condition:

Vital Signs: Frequency of vital signs; input and output recording; neurological or vascular checks. Notify physician if blood pressure <90/60, >160/110; pulse >110; pulse <60; temperature >101.5; urine output <35 cc/h for >2 hours; respiratory rate >30.

Activity: Bed rest or ambulation; bathroom privileges.

Allergies:

Diet: NPO

IV Orders: DS ½ NS at 100 cc/hour.

Oxygen: 6 L/min by nasal canula.

Drains: Foley catheter to closed drainage. Nasogastric tube with instructions for type of suction. Other drains, tubes, dressing changes. Orders for irrigation of tubes as appropriate.

Medications: Antibiotics to be initiated immediately preoperatively; consider additional dose during operation and 1 dose of antibiotic postoperatively to 24 hours post-op. Cefotetan (every 12 hours) for bowel flora, or first generation cephalosporin (Cefazolin) for clean procedures.

Labs and Special X-Rays: Electrolytes, BUN, creatinine, PT/PTT, CBC, platelet count, UA, ABG, pulmonary function test. Chest X-ray (if patient has not had a normal chest x-ray in past 6 months or if >35 yrs old), EKG, (if older than 35 yrs old or if cardiovascular disease). Type and screen for an appropriate number of units of blood.

Preoperative Note

Preoperative Diagnosis:

Procedure Planned:

Type of Anesthesia Planned:

Laboratory Data: Electrolytes, BUN, creatinine, CBC, PT/PTT, UA, EKG, Chest X-ray; type and screen for blood or cross match if indicated; liver function tests, ABG.

Risk Factors: Cardiovascular, pulmonary, hepatic, renal, coagulopathic, nutritional risk factors.

American Surgical Association (ASA) grading (I-V) of surgical risk: 1= normal; 2= mild systemic disease; 3= severe systemic disease; 4= disease with major threat to life; 5= not expected to survive.

Consent: Document explanation to patient of risk and benefits of procedure, and document patient's or guardian's informed consent and understanding of the procedure. Obtain signed consent form.

Allergies:

Major Medical Problems:

Medications:

Special Requirements: Signed blood transfusion consent form; documentation that patient with breast procedure has been given information brochure.

Brief Operative Note

This note should be written in chart immediately after the surgical procedure.

Date of the Procedure:

Preoperative Diagnosis:

Postoperative Diagnosis:

Procedure:

Operative Findings:

Names of Surgeon and Assistant:

Anesthesia: General endotracheal, spinal, epidural, regional or local.

Estimated Blood Loss (EBL):

Fluids and Blood Products Administered During Procedure:

Urine output:

Specimens: Pathology specimens, cultures, blood samples.

Intraoperative X-rays:

Drains:

Operative Report

This report should be dictated at the conclusion of surgical procedure.

Identifying Data: Name of patient, medical record number; name of dictating

physician, date of dictation.

Attending Surgeon and Service:

Date of Procedure:

Preoperative Diagnosis:

Postoperative Diagnosis:

Procedure Performed:

Names of Surgeon and Assistants:

Type of Anesthesia Used:

Estimated Blood Loss (EBL):

Fluid and Blood Products Administered During Operation:

Specimens: Pathology, cultures, blood samples.

Drains and Tubes Placed:

Complications:

Consultations Intraoperatively:

Indications for Surgery: Brief history of patient and indications for surgery.

Findings:

Description of Operation: Position of patient; skin prep and draping; location and types of incisions; details of procedure from beginning to end including description of findings, both normal and abnormal, during surgery. Intraoperative studies or x-rays; hemostatic and closure techniques; dressings applied. Patient's condition and disposition. Needle and sponge counts as reported by operative nurse. Send copies of report to surgeons and referring physicians.

Post-Operative Note

Subjective: Patient's mental status and subjective condition; adequacy of pain control.

Objective:

Vital Signs: Temperature, blood pressure, pulse, respirations.

Physical Exam: Chest and lungs; inspection of wound and surgical dressings; condition of drains; characteristics and volume of output of drains.

Labs: Hematocrit

Assessment: Make an assessment of the patient's overall condition and status of wound.

Plan: Describe post operative and discharge plans.

Post-Operative Orders

1. **Transfer:** From recovery room to surgical ward when stable.
2. **Vital Signs:** q4h, I&O q4h x 24h.
3. **Activity:** Bed rest; ambulate in 6-8 hours if appropriate. Incentive spirometer q1h while awake.
4. **Diet:** NPO x 8h, then sips of water. Advance from clear liquids to regular diet as tolerated.
5. **IV Fluids:** IV D5 LR or D5 ½ NS at 125 cc/h (KCL, 20 mEq/L if indicated), Foley to gravity.
6. **Medications:**
Cefazolin (Ancef) 1 gm IVPB q8h x 3 doses; if indicated for prophylaxis in

clean cases **OR**

Cefotetan 1 gm IV q12h x 2 doses for clean contaminated cases.

Meperidine (Demerol) 50-75 mg IM q3-4h prn pain

Hydroxyzine (Vistaril) 25-50 mg IM q3-4h prn pain **OR**

Prochlorperazine (Compazine) 10 mg IM q4-6h prn nausea or suppository q 4h prn.

7. Laboratory Evaluation: CBC, SMA7, chest x-ray in AM if indicated.

Post-Operative Management

I. Post Op Day #1

- A. Assess pain, lungs, cardiac status; flatulence, bowel movement. Examine for distension, tenderness, bowel sounds; wound drainage, bleeding from incision.
- B. Discontinue IV infusion when taking adequate PO fluids. Discontinue Foley catheter, and use in-and-out catheterization as needed for urinary retention as needed.
- C. Ambulate as tolerated; incentive spirometer, hematocrit and hemoglobin.
- D. Acetaminophen/codeine (Tylenol #3) 1-2 PO q4-6h prn pain.
- E. Colace 100 mg PO bid.
- F. Consider prophylaxis for deep vein thrombosis.

II. Post Op Day #2

- A. If passing gas or if bowel movement, advance to regular diet unless bowel resection.
- B. Laxatives: Dulcolax supp prn, or milk of magnesia, 30 cc PO prn.

III. Post Op Day #3-7

- A. Check pathology report.
- B. Consider removal of staples and placement of steri-strips.
- C. Consider discharge home on appropriate medications; follow up in 1-2 weeks for removal of staples or sutures.
- D. Write discharge orders (including prescriptions) early in AM; arrange for home health care if indicated. Dictate discharge summary and send copy to surgeon and referring physician.

Problem-Oriented Surgical Progress Note

Problem List: Post-operative day number, antibiotic day number, hyperalimentation day number if applicable. List each surgical problem separately (e.g. status-post appendectomy, hypokalemia).

Subjective: Describe how the patient feels in the patient's own words; and give observations about the patient.

Objective: Vital signs; physical exam for each system; thorough examination and description of wound. Condition of dressings; purulent drainage, granulation tissue, erythema; condition of sutures, dehiscence. Amount and color of drainage, laboratory data.

Assessment: Evaluate each numbered problem separately.

Plan: For each numbered problem, discuss any additional orders or surgical

plans. Discuss changes in drug regimen or plans for discharge or transfer. Acknowledge conclusions of consultants.

Discharge Summary

Patient's Name:

Chart Number:

Date of Admission:

Date of Discharge:

Admitting Diagnosis:

Discharge Diagnosis:

Name of Attending or Ward Service:

Surgical Procedures, Diagnostic Tests, Invasive Procedures:

Brief History and Pertinent Physical Examination and Laboratory Data:

Describe the course of the disease up to the time the patient came to the hospital, and describe the physical exam and laboratory data on admission.

Hospital Course: Describe the course of the patient's illness while in the hospital, including evaluation, treatment, outcome of treatment, and medications given while in the hospital.

Discharged Condition: Describe improvement or deterioration in condition.

Disposition: Describe the situation to which the patient will be discharged (home, nursing home), and person who will provide care.

Discharged Medications: List medications and instructions (and write prescriptions).

Discharged Instructions and Follow-up Care: Date of return for follow-up care at clinic; diet, exercise instructions.

Problem List: List all active and past problems.

Copies: Send copies to attending physician, clinic, consultants and referring physician.

Management of the Surgical Patient

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Radiographic Evaluation of Common Interventions

I. Central Intravenous Lines

- A. Central Venous Catheters** should be located above the right atrium in the superior vena cava, and not in a neck vein; the superior vena cava joins right atrium at level of right third costal cartilage. Rule out pneumothorax by checking chest x-ray and verifying that the lung markings extend completely to the rib cages on both sides (an upright, expiratory x-ray may be helpful). Examine for hydropericardium ("water bottle" sign, mediastinal widening). Do not attempt a second central line on the opposite side until chest x-ray shows no pneumothorax.
- B. Pulmonary Artery Catheters** should be located centrally and posteriorly, and not more than 3-5 cm from midline. Do not attempt a second central line on the opposite side until chest x-ray shows no pneumothorax.

II. Pulmonary Tubes

- A. Endotracheal Tubes:** Verify that the tube is located 3 cm below the vocal cords and 4-5 cm above the carina; the tip should be located at the level of aortic arch.
- B. Tracheostomy:** Verify by that the tube is located half the distance from the stoma to the carina; the tube should be parallel to the long axis of the trachea. The tube should be approximately 2/3 of width of trachea, and the cuff should not cause bulging of the trachea walls. Check for subcutaneous air in the neck tissue and for mediastinal widening secondary to air leakage.
- C. Chest Tubes:** A chest tube for pneumothorax drainage should be located at the mid-clavicular line at the level of the third intercostal space. To drain a pulmonary effusion, the tube should be located at the mid to posterior axillary line at the level of the seventh intercostal space. Obtain a chest x-ray to determine position of diaphragm before insertion of chest tube if time permits.
- D. Mechanical Ventilation:** Obtain a chest x-ray to rule out pneumothorax, subcutaneous emphysema, or pneumomediastinum. Infiltrates may diminish or disappear due to increased aeration of the affected lung lobe.

III. Gastrointestinal Tubes

- A. Nasogastric Tubes:** Verify that the tube is in the stomach and not coiled in the esophagus or trachea. The tip of the tube should be in the gastric antrum and should not be near gastroesophageal junction. Insufflate air and listen over the epigastrium to help confirm tube placement.

Blood Component Therapy

- I. **Crystalloids Solutions:** Normal saline, lactated Ringers solution; used for acute volume replacement. 3 cc crystalloid = 1 cc whole blood.
- II. **Colloid Solution Therapy:** Indicated for volume expansion.
 - A. Albumin (5% or 25%): Useful for hypovolemia or to induce diuresis with furosemide in hypervolemic, hypoproteinemic patients. Use salt poor albumin in cirrhosis. Hespan is useful for volume expansion and raising osmotic pressure.
 - B. Purified Protein Fraction (Plasmanate): 83% albumin and 17% globulin; indicated for volume expansion.
 - C. Hetastarch (Hespan): Synthetic colloid; 6% hetastarch in saline. Similar indications as for albumin. Maximum dose 1500 cc per 24 hours.
- III. **Management of Acute Blood Loss and Red Blood Cell Transfusions**
 - A. Control hemorrhage and infuse crystalloids until packed red blood cells are available to replace losses. In trauma, bleeding may require surgical control.
 - B. If crystalloids fail to produce hemodynamic stability after more than 2 liters have been administered, give red blood cells.
 - C. If volume replacement and hemostasis stabilize hemodynamic status, wait for formal type and cross match of blood. In exigent bleeding, administer O negative, low titer blood or type specific (ABO matched), Rh compatible blood which can be obtained rapidly and should be used. Use only in absolute emergency.
- IV. **Guidelines for Blood Transfusion in Anemia**

Consider blood transfusion when hemoglobin is less than 8.0 and hematocrit is less than 24%. If the patient has symptoms of anemia such as chest pain, dyspnea, poor wound healing, mental status changes, earlier transfusion should be provided.
- V. **Blood Component Products**
 - A. **Packed Red Blood Cells (PRBC's):** Each unit provides 250 cc of volume, and each unit should raise hemoglobin by 1 gm/dL and hematocrit by 3%.
 - B. **Platelets:** Indicated for bleeding due to thrombocytopenia or thrombopathy. Each unit should raise platelet count by 5,000-10,000. Usually transfused 8-10 units at a time. Consider platelet transfusion after 8-10 units of blood replacement.
 - C. **Fresh Frozen Plasma (FFP):** Used for bleeding secondary to liver disease, dilutional coagulopathy (from multiple blood transfusions), coagulation factor deficiencies. Requires ABO typing, but not cross matching. Improvement of PT/PTT usually requires 2-3 units. Use one unit of FFP for every four units of PRBC's.
 - D. **Autologous Blood:** Patient donates blood within 35 days of surgery; frozen blood can be stored for up to 2 years. Useful in elective orthopedic, cardiac, and peripheral vascular procedures. Check preoperative hematocrit.

Fluids and Electrolytes

I. Maintenance Fluid Guidelines

70 kg Male: D5 ½ NS with 20 mEq KCL/liter at 125 mL/hr.

II. Pediatric Patients

- A. Use D5 1/4 NS with 20 mEq KCL/liter.
- B. 24 hour water requirement, Kg Method: For the first 10 kg body weight: 100 mL/kg/day PLUS For the second 10 kg body weight: 50 mL/kg/day PLUS For weight above 20 kg: 20 mL/kg/day. Divide by 24 hours to determine hourly rate.

III. Specific Replacement Fluids of Specific Losses

- A. **Gastric (nasogastric tube, emesis):** D5 ½ NS with 20 mEq/liter KCL, replace equal volume of lost fluid q6h.
- B. **Diarrhea:** D5LR with 15 mEq/liter KCL. Provide approximately 1 liter replacement for each 1 kg or 2.2 lb of lost body weight, bicarbonate 45 mEq (½ amp) per liter may be added.
- C. **Bile:** D5LR with 25 mEq/liter (½ amp) of bicarbonate.
- D. **Pancreatic:** D5LR with 50 mEq/liter (1 amp) bicarbonate.
- E. If unusual volume of fluid loss, send aliquot to lab for electrolyte determination.

Evaluation of Postoperative Fever

I. Clinical Evaluation

- A. **History:** Fever ≥ 100.4 -101 F. Determine how many days since operation.
- B. **Differential Diagnosis:** Pneumonia, urinary tract infection, thrombophlebitis, wound infection, drug reaction.
- C. Dysuria, abdominal pain, calf pain. Cough, sputum, headache, stiff neck, joint or back pain.
- D. IV catheter infection (central or peripheral) is an important source of postoperative sepsis.
- E. **Fever Pattern:** Check previous day for fever patterns; spiking fevers indicate abscesses. Continual fevers indicate vascular involvement such as infected prosthetic grafts or septic phlebitis from central IV lines.
- F. Chills or rigors indicate bacteremia, and are usually not associated with atelectasis or drug fevers.
- G. Check for fevers prior to the operation. IV or Foley catheter; alcohol use. Allergies; recent WBC count and differential.

II. Physical Exam

- A. **General:** Temperature. Fever curve, tachycardia, hypotension. Examine wound and all vascular access sites carefully.
- B. **HEENT:** Pharyngeal erythema, neck rigidity.
- C. **Chest:** Rhonchi, crackles, dullness to percussion (pneumonia), murmurs (endocarditis).

- D. **Abdomen:** Masses, liver tenderness, Murphy's sign (right upper quadrant tenderness with inspiration, cholecystitis); ascites. Costovertebral angle or suprapubic tenderness.
 - E. **Extremities:** Infected decubitus ulcers or wounds; IV catheter tenderness (phlebitis); calf tenderness, joint tenderness (septic arthritis). Cellulitis, furuncles, abscesses. Perirectal abscess, buttock abscess from injections.
 - F. **Genitourinary:** Prostate tenderness; rectal flocculence. Cervical discharge, cervical motion tenderness; adnexal tenderness.
- III. **Laboratory Evaluation:** CBC, blood C&S X 2, SMA7, UA, urine C&S; lumbar puncture; blood, urine, sputum, wound cultures; chest X-ray.
- IV. **Differential Diagnosis**
- A. Wound infection, abscesses, intra-abdominal abscess, atelectasis, drug fever, pulmonary emboli, pancreatitis, alcohol withdrawal, deep vein thrombosis, tuberculosis, cystitis, pyelonephritis, osteomyelitis; IV catheter phlebitis, sinusitis, otitis media, upper respiratory infection, pelvic infection, cellulitis; hepatitis, infected decubitus ulcer, peritonitis, endocarditis, diverticulitis, cholangitis, carcinomas.
 - B. **Medications Associated with Fever:** H2 blockers, penicillins, phenytoin, sulfonamides.
- V. **Antibiotics:** Antibiotic therapy should be initiated immediately if there is any possibility of infection.

Sepsis

I. Pathophysiology

- A. Gram-negative organisms are responsible for 50-80% of all cases of septic shock, while 6-24% of cases result from gram-positive organisms. Fulminant parasitic infections, disseminated tuberculosis, and systemic fungal disease are less common causes of sepsis.
- B. The development of gram-negative septic shock is initiated by a lipopolysaccharide component of the gram-negative cell wall, termed endotoxin.
- C. The most common source of gram-negative infection is the genitourinary system.

II. Definitions

- A. **Systemic Inflammatory Response Syndromes (SIRS)** is defined as Two or more of the following:
 1. Temperature $>38^{\circ}\text{C}$ or $<36^{\circ}\text{C}$
 2. Heart rate >90 beats/min
 3. Respiratory rate >20 breaths/min
 4. White blood cell count $>12,000$ or $<4,000$ or $>10\%$ bands
- B. SIRS is commonly caused by infection, but a number of other conditions can cause this syndrome (i.e., trauma, burns, pancreatitis).
- C. **Sepsis** consists of SIRS plus a documented infection.
- D. **Severe Sepsis** consists of Sepsis plus end-organ dysfunction (e.g., hypoxemia, elevated lactate, oliguria, altered mentation).

- E. Septic Shock** is defined as Sepsis with hypotension despite fluid resuscitation plus hypoperfusion abnormalities.
- F. Refractory Shock** is defined as Septic shock that lasts more than 1 hour and does not respond to fluid or pressors.

III. Clinical Stages of Sepsis

- A.** Sepsis begins with an early hyperdynamic vasodilated state ("warm shock") and progresses to a vasoconstricted state with diminished cardiac output ("cold shock"). The earliest signs of sepsis are tachypnea, respiratory alkalosis, and a moderate hyperdynamic state (increased cardiac output and diminished systemic vascular resistance [SVR]) with little change in blood pressure.
- B.** As septic shock progresses, hypotension results from a decrease in SVR which may overwhelm the increase in cardiac output. Hypoperfusion is manifested by oliguria, hypoxemia, and lactic acidosis.
- C.** Later in septic shock the cardiac index decreases as a result of dysfunctional left ventricular performance. Blood pressure remains low and is refractory to attempts at volume expansion. Tissue perfusion is inadequate, and inotropic and vasoactive agents are necessary to maintain blood pressure. Hypotension, cold and clammy skin, hypoxemia, oliguria, and altered mentation occur.

IV. Clinical and Laboratory Manifestations of Sepsis

- A. Fever** is common, although hypothermia may be observed. Absence of fever does not exclude serious infection. 15% of patients may be hypothermic ($T < 97.6^{\circ} \text{F}$) at onset of bacteremia, and 5% never have a temperature above 99.6°F .
- B. Hematologic Abnormalities**
 - 1. Disseminated intravascular coagulopathy (DIC) occurs in 10% of septic patients, but only 2-3% have significant bleeding.
 - 2. This syndrome can appear as hemorrhage, thrombosis, or microangiopathic hemolysis.
 - 3. Laboratory findings include decreased platelets and fibrinogen with elevated prothrombin time, partial thromboplastin time, and fibrin degradation products. Treatment with platelets, coagulation factors, and fibrinogen replacement is reserved for those patients with clinically significant bleeding.
 - 4. **Neutrophilic leukocytosis** with many band forms (left shift) is the most common hematologic change seen in sepsis. Neutropenia is much less common and tends to occur in infants, the elderly, and alcoholics.
- C. Renal Effects**
 - 1. The effect of sepsis on renal function ranges from minimal proteinuria to acute tubular necrosis, renal failure, and death.
 - 2. Diminished effective circulating volume and hypotension cause renal hypoperfusion with ischemia resulting in acute tubular necrosis.
- D. Adult Respiratory Distress Syndrome**
 - 1. Sepsis is the most frequent predisposing factor of ARDS., characterized by increased pulmonary capillary permeability resulting in increased extravascular lung water, a widening of the alveolar-arterial O_2 gradient, and hypoxemia ($\text{PO}_2 < 65 \text{ mm Hg}$) despite efforts at increased oxygenation (40-50% O_2 by mask).

2. Diffuse bilateral infiltrates are present on chest X-ray consistent with pulmonary edema without evidence of volume overload or cardiac failure (pulmonary capillary wedge pressure [PCWP] <18 mm Hg).

E. Cutaneous Manifestations

1. **Ecthyma gangrenosum** is the most notable finding but vesicles, bullae, petechiae, diffuse erythema can occur.
2. Skin lesions should be aspirated, cultured and gram-stained as they may provide early microbiologic confirmation.

V. Clinical Evaluation of Sepsis

A. Blood Cultures: Two to three sets of blood cultures from separate sites are adequate to detect most cases of bacteremia.

B. Gram Stain of Buffy Coat is positive in up to 50% of cases.

C. Hyperglycemia often occurs and may require insulin.

D. Hyperbilirubinemia, minimal transaminase, and alkaline phosphatase elevations are often present due to intrahepatic cholestasis.

E. Laboratory Tests for Serious Infections

Complete blood count, including leukocyte differential and platelet count	Cultures with antibiotic sensitivities
Electrolytes	Blood
Arterial blood gases	Urine
Blood urea nitrogen and creatinine	Endometrium (if endometritis suspected)
Urinalysis	Amniotic fluid (if chorioamnionitis suspected)
INR, partial thromboplastin time, fibrinogen	Wound
Serum lactate	Other sites (eg, sputum, drains)
	Chest X-ray
	Adjunctive imaging studies (eg, computed tomography, magnetic resonance imaging, abdominal X-ray)

VI. Clinical Management of Sepsis

A. Resuscitation

1. Fluid resuscitation can begin with immediate rapid infusion of crystalloid solution (1-2 L of lactated Ringer's or normal saline over 15-20 minutes). Because of the frequent need for large volumes of crystalloid solution and the risk of pulmonary edema, ARDS, and myocardial dysfunction, further hemodynamic therapy should be guided by pulmonary artery catheter (Swan-Ganz catheter) pressures.
2. After the initial fluid bolus, crystalloid solution may be administered at 10 mL/min for 15 minutes. If the PCWP does not increase by 3 mm Hg, the 1 liter fluid bolus is repeated. Optimal PCWP is in the range of 10-15 mm Hg and can usually be achieved with fluid therapy alone.
3. Significant hemodilution may result from the large volumes of fluids; a hemoglobin level of at least 10 g/dL should be maintained.

B. Oxygenation and Ventilation

1. The increased work of breathing and respiratory muscle fatigue during sepsis often necessitates ventilatory support. Early initiation of mechanical ventilation enhances peripheral perfusion.
2. In the patient with sepsis, oxygen therapy should be started if there

is arterial hypoxemia: Oxygen saturation less than 90-92%, or PaO_2 less than 60 mm Hg.

3. Positive end-expiratory pressure is frequently necessary to maintain adequate oxygenation.

C. Vasoactive Drugs

1. Vasoactive drugs are frequently required due to myocardial depression and persistent hypotension.
2. **Dopamine** is the initial drug of choice for improving cardiac function and blood pressure in patients with septic shock. At low doses (1-3 mcg/kg/min), dopamine reacts with dopaminergic receptors, causing vasodilation and increased blood flow in the renal, mesenteric, coronary, and cerebral vasculature. At intermediate doses (5-10 mcg/kg/min), beta-adrenergic effects are predominant, which increase myocardial contractility. At high doses (more than 20 mcg/kg/min), the effect is alpha-adrenergic, and vasoconstriction is seen.
3. If dopamine does not adequately support blood pressure, an attempt should be made to differentiate whether this is due to persistent vasodilation (blood pressure less than 80 mm Hg with SVR less than $1,400 \text{ dynes/s/cm}^2$) or depressed left ventricular function (low left ventricular stroke work index). If a depressed myocardium is the major cause, inotropic therapy (eg, dobutamine or epinephrine) should be started; if persistent vasodilation is the problem, a peripheral vasoconstrictor (phenylephrine or norepinephrine) should be started.

Commonly Used Vasoactive and Inotropic Drugs

Agent	Usual Dose
Dopamine	Renal Perfusion Dose: 1-3 mcg/kg/min (dopaminergic range) Cardiac Inotropic Dose: 5-10 mcg/kg/min (beta-adrenergic) Vasoconstricting Dose: >20 mcg/kg/min (alpha-adrenergic)
Dobutamine	2-20 mcg/kg/min
Norepinephrine	2-8 mcg/min
Phenylephrine	20-200 mcg/min
Epinephrine	1-8 mcg/min

D. Treatment of Infection

1. The choice of drugs should be based on the probable source of infection, gram-stained smears of clinical specimens, the immune status of the patient, and local patterns of bacterial resistance.
2. Aggressive dosing of antibiotics is recommended.

3. **Sepsis:** For initial treatment of life-threatening sepsis in adults, a third-generation cephalosporin (cefotaxime, ceftizoxime or ceftriaxone), ticarcillin/clavulanic acid or imipenem, each together with an aminoglycoside (gentamicin, tobramycin or amikacin) is recommended.
4. **Methicillin-Resistant Staphylococci:** When MRSA is suspected, treatment with vancomycin (with gentamicin) is recommended.
5. **Intra-Abdominal or Pelvic Infections** are likely to involve anaerobes, treatment should include either ticarcillin/clavulanic acid, ampicillin/sulbactam, piperacillin/tazobactam, imipenem, cefoxitin or cefotetan, each with an aminoglycoside or, alternatively, metronidazole or clindamycin, together with an aminoglycoside is necessary.
6. **Biliary Tract Infections:** When the source of bacteremia is thought to be in the **biliary tract**, piperacillin or mezlocillin plus metronidazole, piperacillin/tazobactam, or ampicillin/sulbactam, each with an aminoglycoside, should be used.
7. **Bacterial endocarditis** should be treated with vancomycin and gentamicin.
8. **Neutropenic Patients:** Ceftazidime or imipenem with an aminoglycoside should be used.
9. **Antibiotic-Resistant Gram-Negative Bacilli:** In some hospitals, gram-negative bacilli have become resistant to aminoglycosides, third-generation cephalosporins and aztreonam; these strains may be susceptible to imipenem or ciprofloxacin. *Pseudomonas aeruginosa* strains resistant to gentamicin often are susceptible to amikacin, ceftazidime, imipenem, ciprofloxacin or aztreonam, and sometimes to tobramycin.
10. **Multiple-antibiotic-resistant enterococci**
 - a. Many enterococcal strains are now resistant to ampicillin, gentamicin, and vancomycin. A few vancomycin-resistant enterococci are susceptible to teicoplanin (Targocid). Some strains are susceptible to chloramphenicol, doxycycline or fluoroquinolones.
 - b. Quinupristin/dalfopristin (Synercid) (investigational) is active against most strains of multiple-drug-resistant *Enterococcus faecium* and pneumococci.
 - c. Polymicrobial surgical infections that include antibiotic-resistant enterococci may respond to antibiotics aimed at the other organisms that are present.
11. **Dosages for Antibiotics Used in Sepsis**
 - Cefotaxime (Claforan) 2 gm q4-6h.
 - Ceftizoxime (Cefizox) 2 gm IV q8h.
 - Ceftriaxone (Rocephin) 2 gm IV q12h (max 4 gm/d).
 - Cefoxitin (Mefoxin) 2 gms q6-8h.
 - Cefotetan (Cefotan) 2 gms IV q12h.
 - Ceftazidime (Fortaz) 2 g IV q8h.
 - Ticarcillin/clavulanate (Timentin) 3.1 gm IV q4-6h (200-300 mg/kg/d).
 - Ampicillin/Sulbactam (Unasyn) 3.0 gm IV q6h.
 - Piperacillin/tazobactam (Zosyn) 3.375-4.5 gm IV q6h.
 - Piperacillin, ticarcillin, mezlocillin 3 gms IV q4-6h.
 - Imipenem/cilastatin (Primaxin) 1.0 gm IV q6h (with

gentamicin/tobramycin).

- Gentamicin, tobramycin 5 mg/kg IV qd; or 100-120 mg (1.5-2 mg/kg) IV, then 80 mg IV q8h (3-5 mg/kg/d).
- Amikacin (Amikin) 5.0 mg/kg IV loading dose; then 5 mg/kg IV q8h.
- Vancomycin 500 mg IV q6h, or 1 gm IV q12h.
- Ciprofloxacin (Cipro) 400 mg IV q12h.
- Aztreonam (Azactam) 1-2 gm IV q6-8h; max 8 g/day.
- Metronidazole 500 mg IV q6h.
- Clindamycin 600-900 IV q8h (15-30 mg/kg/d).

- E. The third-generation cephalosporins (ceftriaxone, ceftazidime, cefoperazone, ceftizoxime, cefotaxime), imipenem or aztreonam can be used to treat sepsis caused by many strains of gram-negative bacilli.
- F. Ceftazidime has less activity against gram-positive cocci. Cephalosporins other than ceftazidime and cefoperazone have limited activity against *Pseudomonas aeruginosa*. Imipenem and aztreonam are active against most strains of *P. aeruginosa*, and imipenem is active against anaerobes.
- G. Aztreonam has poor activity against gram-positive bacteria and anaerobes.

VII. Failure to Respond to Appropriate Antibiotics

- A. Failure may suggest an occult or undrained site of infection.
- B. Operative intervention with the removal of infected tissue or drainage of abscesses should be performed. In the postoperative patient, the possibility of a wound infection or abscess should be considered.

Parenteral and Enteral Nutrition

I. Enteral Feeding

- A. **General Measures:** Daily weights, I&O. Head of bed at 30° while enteral feeding and 2 hours after completion. Confirm placement of tube with an X-ray and by instillation of air in tube while auscultating over epigastrium. Do not give enteral feedings in postoperative patients until ileus resolved and gastric emptying is satisfactory. Always check stomach residual fluid before NG feeding. Record bowel movements. Nutrition consult may be useful.
- B. **Enteral Bolus Feeding:** Give 50-100 mL of enteral solution (eg: Osmolite) q3h initially. Increase amount in 50 mL steps to max of 250-300 mL q3-4h; 30 kcal of nonprotein calories/d and 1.5 gm protein/kg/d. Before each feeding measure residual volume, and delay feeding by 1h if >100 mL. Flush tube with 100 cc of water after each bolus.
- C. **Continuous Enteral Infusion** - Initial enteral solution (Osmolite) 30 mL/hr. Measure residual volume q1h x 12h, then tid; hold feeding for 1h if 100 mL. Increase rate by 25-50 mL/hr at 24 hr intervals as tolerated until final rate of 50-100 mL/hr as tolerated. 3 Tablespoons of protein powder (Promix) may be added to each 500 cc of solution. Flush tube with 100 cc water q8h. Do not advance enteral feeding if diarrhea develops.

II. Peripheral Parenteral Supplementation

- 3% amino acid sln (ProCalamine) up to 3 L/d at 125 cc/h **OR**
- Combine 500 mL Amino acid solution (7% or 10%) (Aminosyn) and 500 mL of 20% dextrose and electrolyte additive, and infuse at up to 100 cc/hr in parallel with:
- Intralipid 10% or 20% at 1 mL/min for 15 min (test dose); if no adverse reactions, infuse 500 mL/d at up to 100 mL/hr.
- Draw blood 6h after end of infusion for triglycerides.

III. Central Parenteral Nutrition

- **Indications for TPN:** Severe malnutrition (albumin <3.0 or $>10\%$ loss of body weight), prolonged postoperative ileus, intestinal fistula, pancreatitis. Infuse 40-50 mL/h of amino acid-dextrose solution in the first 24h; increase daily by 40 mL/hr increments until providing 1.3-2 times the basal energy requirement and 1.2-1.7 gm protein/kg/d (formula in appendix).

Standard solution (1 liter) (adjust TPN as needed to maintain normal electrolytes)

Amino Acid (Aminosyn) 7-10%	500 mL
Dextrose 40-70%	500 mL
Sodium	35 mEq
Potassium	36 mEq
Chloride	35 mEq
Calcium	4.5 mEq
Phosphate	9 mMol
Magnesium	8.0 mEq
Acetate	82-104 mEq
Multi-Trace Element Formula	1 mL/d
Regular Insulin (if indicated)	10-60 U/L
Multivitamin(12) (2 amp)	10 mL/d
Vitamin K (in solution, SQ, IM)	10 mg/week
Vitamin B12	1000 mcg/week

Intralipid 20%, 500 mL/d intravenous piggyback, infused in parallel with standard solution at 1 mL/min x 15 min; if no adverse reactions, increase to 100 mL/hr. Serum triglyceride 6h after infusion (maintain <200 mg/dL).

- A. Cyclic TPN** 12h night schedule; Taper continuous infusion in morning by reducing rate to half original rate for 1 hour. Further reduce rate by half for an additional hr; then discontinue. Restart TPN in afternoon. Taper in beginning and end of cycle. Final rate of 185 mL/hr for 12h and 1 hour of taper at each end for total of 2000 mL.

B. Special Medications

- Metoclopramide (Reglan) 10-20 mg PO, IM, IV, or in J tube q6h.
- Cimetidine 300 mg PO tid-qid or 37.5-100 mg/h IV or 300 mg IV q6-8h or in TPN **OR**
- Ranitidine 50 mg IV q6-8h or 150 mg PO bid or in TPN.
- Insulin sliding scale.

C. Symptomatic Medications

- Loperamide (Imodium) 2-4 mg PO/J-tube q6h, max 16 mg/d prn **OR**

- Diphenoxylate/atropine (Lomotil) 1-2 tabs or 5-10 mL (2.5 mg/5 mLs) PO/J-tube q4-6h prn, max 12 tabs/d
- Kaopectate 30 cc PO or in J-tube q8h.

D. Laboratory Evaluation

1. **Baseline** - draw all labs below. CXR, plain film for tube placement
2. **Daily Labs** - SMA7, osmolality, CBC, cholesterol, triglyceride (6 h after infusion).
3. **Weekly Labs** - Cal, phosphorous, SMA-12
4. **Weekly Labs When Indicated** - Protein, Mg, iron, TIBC, transferrin, PT/PTT, zinc, copper, B12, Folate, 24h urine nitrogen and creatinine. Pre-albumin, retinol-binding protein.

Central Venous Catheterization

- I. **Indications for Central Venous Catheter Cannulation:** Monitoring of central venous pressures in shock or heart failure; management of fluid status; administration of total parenteral nutrition. Also used as route for temporary dialysis access and for prolonged antimicrobial or chemotherapy.

II. Location of Catheterization Site

- A. The internal jugular approach is contraindicated in patients with a carotid bruit, stenosis, or aneurysm.
- B. Subclavian approach has increased risk in patients with emphysema or bullae.
- C. The external jugular or internal jugular approach by direct cut-down may be preferable in patients with coagulopathy or thrombocytopenia.
- D. In patients with a chest tube already in place, the catheter should usually be placed on the side with the chest tube.

III. Technique of Insertion for External Jugular Vein

- A. The external jugular vein courses from angle of mandible to behind the middle of clavicle, where it joins with the subclavian vein. Place patient in Trendelenburg's position, and apply digital pressure to the external jugular vein above clavicle to distend vein. Cleanse skin with Betadine iodine solution using sterile technique; inject 1% lidocaine to produce a skin weal.
- B. With an 18-gauge thin wall needle, advance the needle into the vein. Then pass a J-guide wire through the needle; the wire should advance without resistance. Remove the needle, maintaining control over the guide wire at all times. Nick the skin with a No. 11 scalpel blade.
- C. With guide wire in place, pass the central catheter over the wire, and remove the guide wire after the catheter is in place. Cover catheter hub with a finger to prevent air embolization.
- D. Attach a syringe to the catheter hub, and ensure that there is free back-flow of dark venous blood. Attach the catheter to an intravenous infusion at a keep open rate.
- E. Secure the catheter in place with 2-0 silk suture and tape.
- F. Obtain a CXR to confirm position and rule out pneumothorax.
- G. The catheter should be removed and changed within 3-4 days.

IV. Internal Jugular Vein Cannulation: The internal jugular vein is positioned behind the sternocleidomastoid muscle, lateral to the carotid artery. The catheter should be placed at a location at the upper confluence of the two bellies of sternocleidomastoid, at the level of cricoid cartilage.

1. Place the patient in Trendelenburg's position, and turn the patient's head to the contralateral side.
2. Choose a location on the right or left. If all other factors are equal (symmetrical lung function, no chest tubes in place), the right side is preferred because of the direct path to the superior vena cava. Prepare the skin with Betadine solution using sterile technique and drape the area. Infiltrate the skin and deeper tissues with 1% lidocaine.
3. Palpate the carotid artery. Using a 22-gauge scout needle and syringe, direct the needle toward the nipple at a 30 degree angle to the neck. While aspirating, advance the needle until the vein is located and blood back flows into the syringe.
4. Remove the scout needle and advance an 18-gauge, thin wall, catheter-over-needle with an attached syringe along the same path as the scout needle. When back flow of blood is noted into syringe, advance the catheter into the vein. Remove the needle and confirm back flow of blood through the catheter and into the syringe. Remove syringe and cover the catheter hub with a finger to prevent air embolization.
5. With the catheter in position, advance a 0.89 mm x 45 cm guide wire through the catheter. The guide wire should advance easily without resistance.
6. With the guide wire in position, remove the catheter and use a No. 11 scalpel blade to nick the skin.
7. Place central vein catheter over the wire, holding the wire secure at all times. Pass the catheter into the vein, and suture the catheter with O silk suture, tape, and connect to IV infusion at a keep open rate.
8. Obtain a CXR to rule out pneumothorax and confirm position.

V. Subclavian Vein Cannulation

The subclavian vein is located in the angle formed by the medial 1/3 of clavicle and the first rib.

1. Position the patient supine with a rolled towel located longitudinally between the patient's scapulae, and turn the patient's head towards the contralateral side. Prepare the area with Betadine iodine solution, and, using sterile technique, drape the area and infiltrate 1% lidocaine into the skin and tissues.
2. With a 16-gauge catheter-over-needle with syringe attached, puncture the mid-point of the clavicle until the clavicle bone and needle come in contact.
3. Then slowly probe down until the needle slips under the clavicle. Advance the needle slowly towards the vein until the needle enters the vein, and a back flow of venous blood enters the syringe. Remove the syringe, and cover the catheter hub with a finger to prevent air embolization.
4. With the 16-gauge catheter in position, advance a 0.89 mm x 45 cm guide wire through the catheter. The guide wire should advance

easily without resistance.

5. With the guide wire in position, remove the catheter, and use a No. 11 scalpel blade to nick the skin.
6. Pass dilator over the wire.
7. Place the central line catheter over the wire, holding the wire secure at all times. Pass the catheter into the vein, and suture the catheter with 2-0 silk suture, tape the catheter in place and connect to IV infusion.
8. Obtain a CXR to confirm position and rule out pneumothorax.

Pulmonary Artery Catheterization

1. Cannulate a vein using the technique above, such as the subclavian vein or internal jugular.
2. Advance a guide wire through the cannula, and remove the cannula. Nick the skin with number 11 scalpel blade adjacent to the guide wire, and pass a number 8 French introducer over the wire into the vein. Connect introducer to an IV fluid infusion at a keep open rate, and suture introducer in place with 2-0 silk.
3. Pass the proximal end of the pulmonary artery catheter (Swan Ganz) to an assistant for connection to a continuous flush transducer system.
4. Flush the distal and proximal ports with heparin solution, removing all bubbles, and check balloon integrity by inflating 2 cc of air. Check pressure transducer response by moving the distal tip quickly.
5. Pass the catheter through the introducer into the vein, then inflate the balloon, and advance the catheter until the balloon is in or near the right atrium.
6. As a general guideline, the correct distance to the entrance of the right atrium is determined from the site of insertion:
 - Right antecubital fossa: 35-40 cm
 - Left antecubital fossa: 45-50 cm.
 - Right internal jugular vein: 10-15 cm.
 - Subclavian vein: 10 cm.
 - Femoral vein: 35-45 cm.
7. Run a continuous monitoring strip to record pressures as the PA catheter is advanced.
8. Advance the balloon, inflated with 0.8-1.0 cc of air, while monitoring pressures and wave forms. Advance the catheter through the right ventricle into the main pulmonary artery until the catheter enters a distal branch of the pulmonary artery and is stopped by impaction (as evidenced by a pulmonary wedge pressure wave form).
9. Do not advance catheter with balloon deflated, and do not withdraw the catheter with the balloon inflated. After placement, obtain a chest X-ray to insure that the tip of catheter is no farther than 3-5 cm from midline, and no pneumothorax is present.

Normal Pulmonary Artery Catheter Values

Right atrial pressure	1-7 mm Hg
RVP Systolic	15-25 mm Hg
RVP Diastolic	8-15 mm Hg
Pulmonary artery pressure	
PAP Systolic	15-25 mm Hg
PAP Diastolic	8-15 mm Hg
PAP Mean	10-20 mm Hg
PCWP	6-12 mm Hg
Cardiac Output	3.5-5.5 L/min
Cardiac Index	2.0-3.2 L/min/m ²
Systemic Vascular Resistance	800-1200 dyne/sec/cm ²

Venous Cutdown

Procedures

1. Obtain a prepackaged cutdown tray, or a minor procedure tray and instrument tray with a silk suture (3-0, 4-0) and a catheter. This procedure will require gloves, sterile towels/drapes, 4 x 4 gauze sponges, povidone-iodine solution, 5 cc syringe, 25 gauge needle, 1% lidocaine with epinephrine, adhesive tape, scissors, needle holder, hemostat, scalpel and blade, suture. Adequate lighting and assistance should be available before beginning procedure.
2. Apply a tourniquet proximal to the site, and identify the vein. Prep the skin with povidone-iodine solution and drape area. Infiltrate the skin with 1% lidocaine, then incise the skin transversely.
3. Spread the incision long-wise in the direction of the vein with a hemostat and dissect any adherent tissue from the vein. Lift the vein and pass two chromic or silk ties (3-0 or 4-0) behind the vein.
4. Tie off the distal suture, using the upper tie for traction. Make a transverse nick in the vein. If necessary, use a catheter introducer to hold the lumen of the vein open.
5. Make a small stab incision in the skin distal to the main skin incision, and insert a plastic catheter or IV cannula through the incision, then insert it into vein. Tie the proximal suture, and attach IV fluid, release the tourniquet. Suture skin with silk or nylon, and apply dressing.

Arterial Line Placement

Procedure

1. Obtain a 20 gauge, 1½-2 inch catheter-over-needle assembly (Angiocath), arterial line setup (transducer, tubing, pressure bag containing heparinized saline), armboard, sterile dressing, 1% lidocaine, 3 cc syringe, 25 gauge needle, 3-0 silk.
2. The radial artery is used. Use the Allen Test to verify patency of the radial

- artery and adequacy of ulnar artery collaterals. Place the extremity on an armboard with a gauze roll behind the wrist to maintain hyperextension.
3. Prep with povidone-iodine and drape; infiltrate 1% lidocaine using a 25 gauge needle. Choose a site where the artery is most superficial and as distal as possible on the wrist.
 4. Palpate the artery with the left hand, and use other hand to advance a 20 gauge catheter-over-needle into the artery at a 30 degree angle to the skin. When a flash of blood is seen, hold the needle in place and advance the catheter into the artery; occlude the artery with manual pressure while the pressure tubing is connected.
 5. If a needle and guide-wire kits is used, advance the guide wire into the artery, and pass the catheter over the guide-wire.
 6. Suture catheter in place with 3-0 silk and apply dressing.

Cricothyrotomy

I. Needle Cricothyrotomy

- A. Obtain a 12-14 gauge, catheter-over-needle (Angiocath or Jelco), 6-12 mL syringe, 3 mm pediatric endotracheal tube adapter, oxygen tubing, and a high flow oxygen source.
- B. Locate the cricothyroid membrane (notch between the thyroid cartilage and cricoid cartilage).
- C. Cleanse the neck area with povidone-iodine solution, and inject 2% lidocaine with epinephrine (if the patient is awake).
- D. With a 12 or 14 gauge, catheter-over-needle assembly on the syringe, advance needle through the cricothyroid membrane at a 45 degree angle directed inferiorly. Apply back pressure on the syringe until air is aspirated.
- E. Advance the catheter and remove the needle, then attach the hub to a 3 mm endotracheal tube adapter connected to oxygen tubing.
- F. Administer oxygen at 15 liters per minute for 1-2 seconds on, then 4 seconds off; control air flow by using a Y-connector or a hole in the side of the tubing.
- G. The needle cricothyrotomy should be replaced with oral endotracheal intubation as soon as feasible. A needle cricothyrotomy should not be used for more than 45 minutes, since exhalation of CO₂ is inadequate.

II. Surgical Cricothyrotomy

- A. Obtain a #5-#7 tracheostomy tube; tracheostomy tube adapter to connect to bag-mask ventilator; povidone-iodine solution, sterile gauze pads, scalpel handle, and hemostat.
- B. Clean the neck area with povidone-iodine.
- C. Locate the thyroid and cricoid cartilages; the cricothyroid membrane extends between these two cartilages.
- D. Infiltrate the overlying skin with 2% lidocaine with epinephrine if the patient is awake.
- E. Stabilize the thyroid cartilage with the left hand, and make a transverse incision through the skin and subcutaneous tissues overlying the cricothyroid membrane, avoiding the large vessels that are located laterally.
- F. Make a stab incision inferiorly in the cricothyroid membrane with the

point of the blade.

- G.** Insert the knife handle, and rotate the handle 90 degrees to open the hole; or use a hemostat to dilate the opening.
- H.** Gently insert the tube and secure with tape.
- I.** The surgical cricothyrotomy should be replaced with a formal tracheostomy within a few hours.

Trauma

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Trauma Care

- I. **Primary Survey of the Trauma Patient:** Assess airway, breathing, and circulation (ABC's).
 - A = Airway maintenance with cervical spine stabilization.
 - B = Assess breathing, administer assisted ventilations if required; rule out tension pneumothorax.
 - C = Assess circulation and control hemorrhage.
 - D = Assess disability and neurologic status.
 - E = Exposure; completely undress patient; prevent hypothermia.
- II. **Resuscitation Phase**
 - A. Assess adequacy of airway and ventilation, evaluate control of hemorrhage, initiate fluid resuscitation.
 - B. Place a minimum of two large bore IVs. Blood should be drawn for type and cross, hemoglobin or hematocrit, and electrolytes. Initial and follow-up hemoglobin or spun hematocrit should be rapidly determined in the emergency department.
 - C. Administer crystalloids. Initial fluid bolus of 1-2 liters of Ringer's lactate (20 cc/kg in pediatric patients).
 - D. Evaluate for improvement in blood pressure, decrease in pulse rate, improved skin circulation and capillary refill, increase in urine output, and improved mental status.
 - E. If there has been significant hemorrhage, or there has been failure to restore systolic pressure >100mm Hg with crystalloid, transfuse blood.
- III. **Ongoing Assessment and Treatment**
 - A. **Administer cross matched**, type-specific, cross matched, type O negative blood if patient is unstable.
 - B. **Manage coagulopathy** with empiric administration of fresh frozen plasma, 1 unit FFP for every 4 units of packed red blood cells. Transfuse 10 units platelets per 6 units PRBC. If rapid administration of more than 4 units PRBC, consider calcium chloride. Avoid hypothermia.
 - C. **Place a nasogastric tube** for decompression of the stomach, and place a Foley catheter to evaluate urine output (caution if facial fracture or unstable cervical spine).
 - D. **Monitor adequacy** of resuscitation, looking for improvement in physiologic parameters such as heart rate, systolic pressure, ventilatory rate, distal perfusion and capillary refill, pulse oximetry.
 - E. **Reassess ABC's** prior to beginning secondary survey.
- IV. **Secondary Survey**
 - A. Obtain an abbreviated history including allergies, medications, past illness, Last meal, Event/mechanism= AMPLE history.

- B. Evaluate the completely undressed patient, front and back and from head to toe, and thoroughly evaluate each system (head and neck, chest, abdomen, perineum, musculo-skeletal, vascular, neurologic). Check the patient's Glasgow coma scale (GCS) score.
- C. Obtain appropriate X-rays (chest, cervical spine, pelvis) and other procedures (peritoneal lavage, CT scan). Do not send an unstable patient to the radiology department.
- D. **Laboratory Studies:** Send type and cross for 6 units or more of packed red blood cells; complete blood count, platelet count, electrolytes, creatinine, BUN, glucose, calcium, PT/PTT; ethanol level, pregnancy test, arterial blood gases; UA, urine and serum toxicology screens. Liver function tests, amylase.
- E. **Re-evaluation:** constantly re-evaluate for current status/response to therapy.

V. Definitive Care Phase

- A. After preliminary identification of injuries, begin definitive care management of life threatening emergencies and obtaining urgently required studies.
- B. Reassessment and simultaneous treatment should continue. The most immediate life threatening problems should be treated first: ABC's followed by abdominal trauma; neurologic or head trauma; massive blood loss via pelvic or extremity fractures.

Abdominal Trauma

- A. Maintain airway, breathing, and circulatory support. Rapid exsanguinating injuries take precedence over other injuries, including head injuries.
- B. **Initial Stabilization:** Control external bleeding with direct external pressure.
- C. Place two number 14 or 16 gauge intravenous lines, type and cross for packed blood cells.
- D. In an emergency (with insufficient time to cross match), give type O-negative blood.
- E. For hypotensive patients, give an initial fluid challenge of 2 L of Ringer's lactate solution over 5-10 min or 20 mL/kg in children over 5-10 min.
- F. **Assess response to initial fluid challenge** by checking blood pressure and heart rate.
- G. **Patients who respond with only a transient increase** in blood pressure should be rechallenged with Ringer's lactate or blood transfusion; blood loss may be continuing.
- H. Patients who have no response to initial fluid challenge may have either extensive blood loss or continued bleeding, and must be given rapid blood transfusion. The site of bleeding must be identified (chest, abdomen, extremities, pelvis) and surgical intervention should be initiated.
- I. **Manage coagulopathy** with empiric administration of fresh frozen plasma, 1 unit FFP for every 4 units of packed red blood cells. Transfuse 10 units platelets per 6 units PRBC.
- J. Keep the patient, ventilator circuit, and infused fluids warmed.

Hypothermia often contributes to coagulopathy. Consider calcium infusion if rapid infusion of greater than 4 U PRBC.

- K. If peritoneal or visceral penetration is suspected**, broad spectrum beta-lactam (cefoxitin, cefotetan, Timentin or Zosyn) antibiotic coverage is necessary, but patient should be taken immediately to the operating room. Continue for 48 hours if bowel injury.

Penetrating Abdominal Trauma

I. Gun Shot Wounds

- A.** All abdominal gun shot wounds require exploratory laparotomy.
- B.** Tangential wounds that do not penetrate the peritoneal cavity may be assessed by peritoneal lavage or laparoscopy if wound is located on the anterior abdominal wall.

II. Stab Wounds and Other Penetrating Abdominal Trauma

- A.** Exploratory laparotomy is required if acute abdomen or signs of peritoneal injury, shock, hypotension, upper or lower GI bleeding, evisceration or pneumoperitoneum.
- B.** If patient is stable and the abdominal fascia is involved, or penetrated on local exploration, perform diagnostic peritoneal lavage. If lavage is positive, perform exploratory laparotomy. (RBC >1000/mL, WBC >50). The threshold for acceptable cell count is much lower after penetrating abdominal injuries than after blunt abdominal trauma.
- C.** Consider tetanus prophylaxis if indicated.

III. Stab Wounds and Other Sharp Trauma With No Involvement or Penetration of Fascia

- A.** These patients should be observed for 24 hours, and local wound care and antibiotics should be administered.
- B.** Consider tetanus prophylaxis if indicated.

Blunt Abdominal Trauma

- I. If Acute Abdomen or Pneumoperitoneum On X-ray:** Perform exploratory laparotomy.

II. If Non-Acute Abdomen

- A.** Perform diagnostic peritoneal lavage or CT scan if possible internal injury.
- B.** If patient is not stable (SBP <100, HR >100, decreasing hemoglobin) and abdominal injury is possible, DPL should be done rather than CT scan. If lavage is positive, laparotomy is required.
- C.** If a CT scan shows only minor isolated splenic or liver injury, and patient remains stable, the patient may be observed in the ICU. Other injuries mandate laparotomy.
- D.** CT scan sensitivity is influenced by the experience of the person reading the scan. CT scan is not sensitive for intestinal or diaphragmatic injury.
- E.** In stable patients who do not receive exploratory laparotomy, serial

abdominal exams should be performed. If significant findings of tenderness or peritoneal signs, the patient should be evaluated by DPL, CT, or laparotomy.

- F. If significant head injury, intoxication, or distracting injury (eg, multiple rib fractures, pelvic fracture, extremity fracture), the abdominal exam is unreliable, and the patient must be evaluated by DPL or CT scan.
- G. If patient is to undergo a prolonged orthopedic or neurosurgical procedure, the abdomen should be evaluated with DPL or CT before the procedure; if subdural or epidural hematoma mandates immediate operation, a DPL can be done in the operating room.
- H. DPL will not detect reroperitoneal injuries. Previous abdominal surgery is a relative contraindication to DPL; if DPL is indicated, it should be done by open rather than Seldinger technique. Morbid obesity and advanced cirrhosis are also relative contraindications to DPL.
- I. **Criteria for a Positive Peritoneal Lavage**
 - 1. Gross blood; red blood cell count $>100,000$ cells/mm³; white blood cell count >500 cells/mm³.
 - 2. Presence of food particles, bile, feces, or bacteria on Gram stain.
 - 3. Exit of lavage fluid via a chest tube or bladder catheter.
 - 4. Amylase ≥ 20 IU/L; alkaline phosphatase ≥ 3 IU.

Diagnostic Peritoneal Lavage

- 1. Insert a nasogastric tube and Foley catheter to decompress the stomach and the bladder. Restrain or sedate patient if necessary. Prep and drape the periumbilical region with Betadine solution and sterile towels. Select a site above or below umbilicus. If the patient has a pelvic fracture or if pregnant, the site must be above the umbilicus.
- 2. Infiltrate the skin and subcutaneous tissue with 1% lidocaine with epinephrine. Incise the skin with a 1-5 cm vertical incision through the subcutaneous tissue down to fascia.
- 3. Use a No. 11 scalpel blade to make a stab incision 2-3 mm in length into the fascia.
- 4. Apply traction to both sides of fascial incision with towel clips, and have an assistant apply strong upward traction on clips.
- 5. Disect bluntly with a small hemostat to peritoneum, then grasp and incise the peritoneum, and introduce a lavage catheter into the pelvis.
- 6. Aspirate with a 12 cc syringe. If 6 cc of blood is returned, the lavage should be considered "grossly positive", which mandates an immediate laparotomy. If the aspirate is negative, instill 1 liter of Ringer's lactate or saline from a pressure bag. Periodically agitate the abdomen. When only a small amount of fluid remains in the bag, drop bag to the floor, and drain the fluid by siphon action.
- 7. During the procedure keep a sponge packed in the wound and hold catheter in place. After at least 300 cc of fluid have been removed, clamp the tubing and withdraw the catheter.
- 8. Close the fascial defect with heavy absorbable suture, and staple the skin.

Head Trauma

I. Initial Management of Head Trauma

- A. Maintain airway and control cervical spine. Management of Airway Breathing and Circulation must take priority (follow the ABC's of basic cardiac life support).
- B. A cervical spine injury should be considered to be present in any patient with multisystem trauma, especially injury involving the area above the clavicles. A normal neurologic examination does not rule out a cervical spine injury.
- C. Intravenous resuscitation solutions should be isotonic (lactated Ringers or normal saline).
- D. Make an initial assessment of the patient.
- E. Perform a mini-neurologic examination as soon as possible and repeat frequently (GCS, pupils, motor/lateralizing signs).
- F. If altered mental status, give 50 mLs of 50% dextrose, thiamine 100 mg, and naloxone (Narcan) 0.4-2.0 mg, intravenously.
- G. Take a complete history, including the mechanism of injury, past medical history, drug intake.

H. Glasgow Coma Scale Assessment of Level of Consciousness Points

1. Eye Opening

spontaneous	4
to speech	3
to pain	2
none	1

2. Verbal Response

oriented	5
confused	4
inappropriate words	3
incomprehensible sounds	2
none	1

3. Best Motor Response

obeys	6
localities	5
withdraws	4
flexion	3
extension	2
none	1

- I. Assess for lateralizing extremity weakness.
- J. Assess pupillary function.
- K. Examine skull for clinically detectable depressed, skull fracture; Battle's sign (blood in the ear canal or ecchymosis over mastoid process); Raccoon's eyes (periorbital ecchymosis), rhinorrhea. The patient requires admission if any of these signs are present.
- L. Do not place a nasogastric or nasotracheal tube if a cribriform plate fracture is possible.

II. Secondary Management of Head Trauma

- A. All patients need admission and 24 hours of serial neuro-exams unless GCS is 15 and only brief amnesia of events, without loss of consciousness. Such patients may be discharged with instructions if

reliable observation ensured.

- B. If GCS 14 or less, or if loss of consciousness for more than a few seconds, a head CT scan should be obtained.
- C. If the mechanism of injury was significantly violent (rollover of vehicle), or if massive upper torso trauma or any lateralizing neuro deficits, a head CT should be obtained.
- D. **If GCS is less than 8 or if unequal pupils, lateralizing deficits, or Open Head Injury:** There is a high probability of mass effect (subdural, epidural, or intracerebral bleed) or diffuse axonal injury. This patient requires ICU admission after obtaining a CT of head and a neurosurgical consultation. Emergency intubation is required for airway control and hyperventilation to PCO_2 30-32 mm Hg until assessment of intracerebral pressure by head CT and/or ICP monitoring. Intubation is done orotracheally with on-line-traction (do not hyperextend neck).

III. Ongoing Management of Head Trauma

- A. Continually reassess ABC's, ECG, systolic, blood pressure, HR, pulse oximeter. Serial Hgb or Hct should be obtained.
- B. Isolated head injuries do not cause hypotension, alternate etiologies must be vigorously sought.
- C. Draw blood for CBC, PT, PTT, Chem 18; type and cross matching; toxicology screen, serum osmolality.
- D. Give stress ulcer prophylaxis with H_2 -blockers (ranitidine, cimetidine), or sucralfate.
- E. Avoid administration of sedative-hypnotics, narcotics, or neuromuscular blockers unless directed by neurosurgeon.
- F. Control hypothermia aggressively with warming of IV fluids, blood, ventilator circuit, and with warming blankets.
- G. Clean and repair open head wounds.
- H. Give Tetanus prophylaxis with 0.5 cc tetanus toxoid IM, with or without tetanus Ig 250 IU. IM as indicated.
- I. **Antibiotics:** Penetrating head and neck injuries should receive Ancef, abdominal trauma injuries should receive a 2nd generation cephalosporin, open extremity fracture injuries should receive Ancef plus Gentamicin.

Pneumothorax

I. Management of Pneumothorax

- A. **Small Primary Spontaneous Pneumothorax (<10-15%) (no underlying pulmonary disease, isolated injury, and not dyspneic)**
 - 1. Observe for 24 hours. Treat with high flow oxygen. Repeat exams frequently. Pulse oximeter monitoring.
 - 2. Repeat chest X-ray in 4 hours. If the pneumothorax does not increase in size, and the patient remains asymptomatic after 24 hours, discharge home. The patient should be instructed to rest and curtail all strenuous activities. Return if increase in dyspnea or recurrence of chest pain.
- B. **Traumatic Pneumothorax associated with a penetrating injury with hemothorax, or traumatic pneumothorax requiring positive pressure ventilation, or with tension pneumothorax**

1. Give high flow oxygen, and immediately insert a chest tube. Provide aggressive hemodynamic and respiratory resuscitation as indicated.
2. Do not delay the management of a possible tension pneumothorax until radiographic confirmation; needle thorocostomy may provide temporizing measure, but insert a chest tube as soon as possible.

II. Technique of Chest Tube Insertion

- A.** Place patient in the supine position, with involved side elevated 10-20 degrees, and abduct arm at 90 degrees. The usual site is between the mid-axillary and anterior axillary line at the level of the fourth intercostal space (nipple line). Alternatively, the second or third intercostal space, in the mid-clavicular line may be used for pneumothorax drainage alone (not useful for hemothorax drainage, and should be avoided in the emergency setting).
- B.** Cleanse skin with Betadine iodine solution and drape the field. Determine the intrathoracic tube distance (lateral chest wall to the apices), and mark length of tube with a clamp.
- C.** Infiltrate 1% lidocaine into the skin, subcutaneous tissues, intercostal muscles, periosteum, and pleura using a 25-gauge needle. Use a scalpel to make a transverse skin incision, 2 centimeters wide, located over the rib just inferior to the interspace where the tube will penetrate the chest wall.
- D.** Use a Kelly clamp to bluntly dissect a subcutaneous tunnel from the skin incision, extending just over the superior margin of the rib. Avoid the nerve, artery, and vein located just below each rib.
- E.** Bluntly dissect over the rib and penetrate the pleura with the clamp, and open the pleura 1 centimeter.
- F.** With a gloved finger, explore the subcutaneous tunnel, and palpate the lung medially. Exclude possible abdominal penetration, and verify correct location within the pleural space; use finger to remove any local pleural adhesions.
- G.** Use the Kelly clamp to grasp the tip of the thoracostomy tube (36 F, Argyle, internal diameter 12 mm), and direct it into the pleural space in a posterior, superior direction for pneumothorax evacuation. Direct tube inferiorly for pleural fluid removal. Guide the tube into the pleural space until the last hole is inside the pleural space and not inside the subcutaneous tissue.
- H.** Attach the tube to 20 cm H₂O of suction. Suture the tube to the skin of the chest wall using O silk. An untied, vertical, mattress suture may be placed in order to close the skin after removal of chest tube.
- I.** Apply Vaseline gauze, 4 x 4 gauze sponges, and elastic tape. Obtain a chest X-ray to verify correct tube placement and to evaluate re-expansion of the lung.
- J.** Indications for Thoracotomy After Trauma
 1. >1000 mL blood from chest tube on insertion
 2. >200 mL blood/hour from chest tube thereafter
 3. Massive air leak such that lung will not re-expand, despite properly placed and functioning chest tube.

Tension Pneumothorax

I. Clinical Signs

- A. Severe hemodynamic and respiratory compromise; contralaterally deviated trachea; decreased or absent breath sounds on the involved side.
- B. Hyperresonance to percussion on the affected side; jugular venous distention, asymmetrical chest wall motion with respiration.

II. Radiographic Findings

- A. Flattening or inversion of the ipsilateral hemidiaphragm; contralateral shifting of the mediastinum.
- B. Flattening of the cardiomedastinal contour, and spreading of the ribs on the ipsilateral side.
- C. Loss of lung markings of ipsilateral side.

III. Acute Management of Tension Pneumothorax

- A. Place a chest tube immediately as described above. A temporary large-bore IV catheter may be inserted into the pleural space, at the level of the second intercostal space at the mid-clavicular line, until the chest tube is placed.
- B. Insert 2 large-bore intravenous lines. Intubation and central venous pressure monitoring and an arterial line may also be indicated.
- C. Draw blood for CBC, PT, PTT, type and cross-matching.

Flail Chest

I. Clinical Evaluation

- A. Usually secondary to severe, blunt, chest injury with multiple rib fractures of adjacent ribs, with more than one fracture per rib. The fractured ribs allow a rib segment without bony continuity with the rest of the chest wall, to move freely during breathing.
- B. Arterial blood gases should be measured if respiratory compromise is significant. If fracture of left, lower, rib cage, rule out splenic injury with CT scan.

II. Management of Flail Chest

- A. Aggressive pulmonary suctioning and close observation for any signs of respiratory insufficiency or hypoxemia.
- B. Endotracheal intubation and positive-pressure ventilation is indicated for significant cases of flail chest if blood gas analysis reveals inadequate ventilation or oxygenation, or in the multiply injured, severely compromised patient.
- C. Associated injuries such as pneumothorax and hemothorax should be treated with tube thoracostomy.
- D. Pain control with an epidural or intercostal blockade may eliminate the need for intubation. Intubation is required if there are significant injuries with massive pulmonary contusion or poor pulmonary reserve.
- E. If mechanical ventilation is required, the ventilator should be set to assist control mode to allow the flail segment at rest for several days.

Thereafter, a trial of low rate, intermittent, mandatory ventilation may be attempted to check for return of flail, prior to attempting extubation.

Massive Hemothorax

I. Clinical Evaluation

- A. Defined as >1500 mL blood lost into the thoracic cavity; most commonly secondary to penetrating injuries.
- B. Absence of breath sounds and dullness to percussion on the ipsilateral side; signs of hypovolemic shock.

II. Management

- A. Simultaneously restore volume deficit and decompress chest cavity with a chest tube. Consult cardiothoracic surgeon as soon as possible.
- B. Place two large-bore intravenous lines or a central venous line.
- C. Insert a chest tube as described previously, except place the site of insertion at the level of the fifth or sixth intercostal space, along the midaxillary line, ipsilateral to the hemothorax. The chest tube should be inserted in a location away from the injury, and it should not be inserted through the injury site.
- D. Clean and close the penetrating wound, and cover it with Vaseline impregnated gauze to decrease the likelihood of tension pneumothorax.
- E. A thoracotomy is indicated if blood loss continues through the chest tube. (>200 mL/hr for 2-3 consecutive hours). If the site of wound penetration is medial to the nipple anteriorly or medial to the scapula posteriorly, this represents a higher probability for injury to the myocardium and the great vessels. If the wound is below the fourth intercostal space, abdominal injury is possible and must be assessed.
- F. Consider tetanus prophylaxis and empirical antibiotic coverage.

Cardiac Tamponade

I. Clinical Evaluation

- A. Cardiac Tamponade most commonly secondary to penetrating injuries. Cardiac Tamponade can also occur when a central line penetrates the wall of the right atrium.
- B. Cardiac Tamponade is often manifested by Beck's Triad of Venous pressure elevation, drop in the arterial pressure, muffled heart sounds.
- C. **Other Signs Include:** Signs and symptoms of hypovolemic shock; pulseless electrical activity (electromechanical dissociation); decreased voltage on ECG, enlarged cardiac silhouette on CXR.
- D. **Kussmaul's Sign:** Rise in venous pressure with inspiration. Pulsus paradoxus or elevated venous pressure may be absent when associated with hypovolemia.

II. Management

- A. Pericardiocentesis or pericardial window is indicated if patient is unresponsive to fluid resuscitation measures for hypovolemic shock, and tamponade is suspected.
- B. All patients who have a positive pericardiocentesis (recovery of non-

clotting blood) due to trauma, require open thoracotomy with inspection of the myocardium and the great vessels. Immediately consult cardiothoracic surgery.

- C. Temporarily with IVF or blood on way to OR.
- D. Consider other causes of hemodynamic instability or electromechanical dissociation that may mimic cardiac tamponade, such as tension pneumothorax, massive pulmonary embolism, or hypovolemic shock.
- E. Subxiphoid pericardial window is as rapid and safer than pericardiocentesis if equipment and experienced surgical personnel are available. If not, pericardiocentesis should be done.

Pericardiocentesis

I. Management

- A. Infusion of Ringer's lactate, crystalloid, colloid, and/or blood may provide a temporizing measure.
- B. Protect airway; unstable patients require intubation. If the patient can be stabilized, pericardiocentesis or a subxiphoid pericardial window should be accomplished in the operating room.
- C. Place patient in supine position. Cleanse and drape peri-xiphoid area, and infiltrate lidocaine 1% with epinephrine into the skin and deep tissues (if time permits).
- D. Attach a long, large bore (12-18 cm, 16-18 gauge), short bevel, cardiac needle to a 50 cc syringe with a 3-way stop cock. Attach a V-lead of the ECG to the metal of the needle with an alligator clip if time permits.
- E. Advance the needle just below costal margin, immediately to the left and inferior to the xiphoid process. Apply suction to the syringe while advancing the needle, slowly, at a 45 degree angle towards the mid point of the left clavicle.
- F. As the needle penetrates the pericardium, resistance will be felt, and a characteristic "popping" sensation will be noted.
- G. Monitor the ECG for ST segment elevation (indicating ventricular heart muscle contact); or PR segment elevation (indicating atrial epicardial contact). After the needle comes in contact with the epicardium, withdraw the needle slightly. Ectopic ventricular beats are associated with cardiac penetration.
- H. Aspirate as much blood as possible. Blood from the pericardial space usually will not clot. Blood, inadvertently, drawn from inside the ventricles or atrium usually will clot. If fluid is not obtained, withdraw and redirect the needle in a more medial direction.
- I. Stabilize the needle by attaching a hemostat or Kelly clamp.
- J. Consider emergency thoracotomy to determine the cause of hemopericardium if the patient develops profound hypotension (<70 mm Hg), otherwise definite treatment is best achieved in the operating room.
- K. If the patient does not improve, consider other problems that may resemble tamponade, such as tension pneumothorax, pulmonary embolism, or shock secondary to massive hemothorax.

Other Life-Threatening Trauma Emergencies

I. Cardiac Contusions

- A. Arrhythmias are the most common consequence of cardiac contusions, but pump failure can also occur.
- B. **Treatment:** The patient should receive cardiac monitoring for 4 hours and treatment of arrhythmias. If pump failure is suspected, assess cardiac function with an echocardiogram and/or Swan Ganz, and provide inotropic support.

II. Pulmonary Contusions

- A. Pulmonary contusions are the most common potentially lethal chest injuries following blunt chest trauma. Respiratory failure and hypoxemia may develop gradually over several hours. A continuing index of suspicion is required to make the diagnosis. The clinical severity of hypoxia does not correlate well with chest x-ray.
- B. If pulmonary compromise is mild and the patient is not multiply injured, selected patients can be managed without intubation.
- C. Treatment of significant contusions, especially in multiply injured patients, consists of intubation, positive pressure ventilation, and PEEP.

III. Traumatic Aortic Transection (deceleration)

- A. Diagnosis requires a high index of suspicion in patients with an appropriate mechanism of injury.
- B. The chest x-ray may show a widened mediastinum, obscured aortic knob, or left pleural cap. The diagnostic gold standard remains aortogram, although transesophageal echocardiogram and helical CT scan are gaining acceptance.

IV. Pelvic Fracture

- A. Fracture of the pelvis can produce exsanguinating hemorrhage. Diagnosis is by physical exam, plain x-ray films, and CT scan.
- B. Hemorrhage is difficult or impossible to control at laparotomy. Most bleeding is venous, and is best treated acutely by external fixation of the pelvis. Arterial bleeding sometimes occurs, and requires angiographic embolization.
- C. Pelvic fractures are often associated with abdominal injury. Diagnostic peritoneal lavage can be utilized to establish the presence of internal hemorrhage. Associated bladder or urethral injuries are also common.

- V. **Extremity Fractures:** Femur fractures, especially bilateral, can cause hemorrhage of several units of blood which can be life threatening, especially in elderly or multiply injured patients.

VI. Traumatic Esophageal Injuries

- A. **Clinical Evaluation:** Esophageal injuries are usually caused by penetrating chest injuries or severe blunt trauma to the abdomen, or by a nasogastric tube or endoscopy, or by repeated vomiting (Boerhaave's syndrome).
- B. After rupture, esophageal contents leak into the mediastinum, followed by immediate or delayed rupture into the pleural space (usually on left), with resulting empyema.

- C. A high index of suspicion is required, particularly in transthoracic penetrating injuries. Transmediastinal penetrating injuries mandate a workup for great vessel, tracheobronchial, and esophageal injuries.
- D. **Treatment of Esophageal Injuries**
 1. Surgical therapy consists of primary repair, or esophageal diversion in the neck and a gastrostomy. Perforated tumors should be resected.
 2. Start empiric broad spectrum antibiotic therapy as soon as possible.

Burns

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I. Immediate Management of Burns

- A. Stop the burning process by removing hot clothing, cool the wound with tepid water or saline (but avoid hypothermia), irrigate chemical burns; remove victim from electrical source.
- B. Establish airway and administer oxygen, establish intravenous line.
- C. **Airway maintenance.**
 1. The airway takes first priority. Smoke inhalation should be suspected if fire occurred in a closed space or if there are thermal injuries to the face, nares, or upper torso. Examine nasal passages for soot, swelling or burns of the nasal vibrissae. Intubation is indicated if there is any evidence of an upper airway burn.
 2. Blood gases, including carboxyhemoglobin, should be determined. If the patient has extreme air hunger or is in critical condition, endotracheal intubation is indicated.
 3. Once intubated, leave endotracheal tube in place at least 72 hours. The tube must be well secured with a tie around the head. Tape will not be adequate to hold tube.

D. Assess Percentage of Burn Area

Head	9%	Posterior Leg	9%
Anterior Trunk	1%	Arm	9%
Posterior Trunk	1	8Genitalia	1%
	%		
Anterior Leg	9%		

- E. Burns involving more than 15% of the total body surface area in adults, or more than 10% in children, should be transferred to a burn center. Patients with other severe injuries, extremes of age or inhalation injury should also be admitted to a burn center.
- F. **Fluid Resuscitation**
 1. Place at least two 16 gauge IV catheters, arterial line, and consider a central line.
 2. A urinary catheter should be inserted.
 3. Administer crystalloid solution (Ringer's lactate) during the first 24 hours. The amount of crystalloid solution administered should be

determined by blood pressure, pulse, and urinary output; formulas are useful guidelines but should not be adhered to rigidly if circumstances dictate otherwise.

4. The Parkland formula may be used as a guide to fluid requirements: First 24 hours--balanced salt solution (lactated Ringer's injection), 4 mL/kg/%burn; give half of this during the first 8 hours and the other half over the next 16 hours. Colloid 0.3-0.5 cc/% burn/kg body weight may also be given. Fluid requirements should be modified for each patient based on their perfusion.
5. Monitoring of fluid resuscitation: Fluid orders should be adjusted hourly. Urine output remains the best means of monitoring resuscitation. 0.5 mL/kg/hour is the acceptable minimal output in adults; 1 mL/kg/hour in children. Peripheral perfusion and sensorium should also be followed.
6. Children may require more free water than older patients; this can be monitored by frequent determinations of serum osmolality or serum sodium. Urine specific gravity can also be used to assess the amount of free water needed.

II. Assessment of Burns

- A. **First Degree Burn:** Involve epidermis only (sunburn); blanching erythema; very painful; not included in the estimation of extent of burn injury.
- B. **Second Degree Burn:** Destruction of epidermis with extension into underlying dermis.
- C. **Third Degree Burn:** Involve destruction of epidermis and dermis, with extension into subcutaneous tissue; painless.
- D. **Fourth Degree Burn:** Extend into subcutaneous tissue, muscle, fascia, or bone.

III. Medications

- A. **Pain Control:** Initially IV morphine 0.1 mg/kg; or meperidine 1-2 mg/kg should be given to relieve all pain. Give pain medications before severe pain occurs, on an hourly basis (not "as needed").
- B. Diazepam 5-10 mg IV doses may be given after circulation has stabilized.
- C. Intravenous H₂ blockers should be routinely administered.
- D. **Antibiotics:** Not given prophylactically. If a patient becomes febrile, hypotensive, anuric, hypothermic, or deteriorates despite adequate fluids, the most likely diagnosis is sepsis. After taking appropriate cultures, broad spectrum antibiotics should be given.
- E. Tetanus toxoid (0.5 cc) should be administered to all patients. If the wound is >50% of the body surface area, 250 units of tetanus immune globulin (human) should also be administered.

IV. Burn Wound Management

- A. Initial examination and debridement can occur during fluid resuscitation.
- B. The burn should be initially thoroughly cleaned with sterile saline and antibacterial solution (normal saline: iodoform in a 2:1 dilution). Avoid vigorous scrubbing. Use sterile scissors to remove the charred epithelium, surface debris; leave unbroken blisters intact. Rinse with copious amounts of normal saline.
- C. After cleaning of burns, they should be dressed with occlusive

dressings combined with antibacterial cream (usually silver sulfadiazine). The ointment or cream may be applied to the patient or to the gauze. The cream should then be covered with elastic gauze netting.

- D. Silver sulfadiazine may cause transitory leukopenia, due to margination of white cells. This leukopenia is benign and does not necessitate discontinuing the drug.
- E. Burn areas that are full thickness or deep partial thickness should be excised and grafted during the first few post-burn days.
- F. Burns that will heal in 14-21 days are best treated nonsurgically.
- G. Patients should be bathed daily at which time the dressings or cream are washed off. Debridement of all loose tissues is performed during the bath.

H. Extremity Burns

1. Circumferential burns and subsequent edema may impair circulation. Impaired circulation must be relieved by escharotomy.
2. Prior to escharotomy, tissue pressure should be monitored clinically, or by tissue pressure measurements until pressure measurements have been less than 40 mmHg for more than 72 hours.
3. Hand burns should be splinted in the anti-claw position and physical therapy should be initiated immediately.

V. Ongoing Patient Care

- A. **Nutrition:** Feeding via a nasogastric or nasoduodenal tube should be initiated immediately.
- B. **Psychosocial Issues:** The presence of post-traumatic stress disorder may require psychotherapy. Support groups may be helpful. For children, the possibility of child abuse must be ruled out.
- C. **Discharge Planning:** Prior to discharge, the pain should be adequately controlled with oral medications, and arrangements for ongoing wound care and therapy should be made.

Pulmonology

Charles F. Chandler, M.D.

Airway Management and Intubation

I. Oral Tracheal Intubation

ETT Size (interior diameter):

Women	7.0 - 8.0 mm
Men	7.0 -9.0 mm.

- A. Prepare functioning suction apparatus, and have bag and mask apparatus set-up with 100% oxygen.
- B. **Sedation And/or Paralysis:**
 - Fentanyl (Sublimaze) 50 mcg increments IV (1 mcg/kg) **with:**
 - Midazolam hydrochloride (Versed) 1 mg IV q2-3 min, max 0.1-0.15 mg/kg **OR**
 - Etomidate (Amidate) 0.2-0.3 mg/kg IV **OR**
 - Propofol (Diprivan) 1-2 mg/kg (can have cardiac depressant effect)**followed by:**
 - Succinylcholine (Anectine) 1 mg/kg (may cause hyperkalemia). **OR**
 - Atracurium 0.5 mg/kg - shorter acting than succinylcholine, but may cause histamine release/hypotension.**Note:** These drugs may cause vomiting; therefore, apply cricoid cartilage pressure during intubation (Sellick maneuver).
- C. Position patient's head in the "sniffing" position with neck hyperextended.
- D. Hyperventilate patient with bag mask apparatus and 100% oxygen, until patient pulse oximeter >95% saturated.
- E. Hold Endoscope handle with left hand, and use right hand to open the patient's mouth. Insert along right side of mouth to the base of tongue, and push tongue to left. Advance to the vallecula (superior to epiglottis) if using curved blade, and lift anteriorly, being careful not to exert pressure on the teeth. If using a straight blade, place beneath the epiglottis and lift anteriorly.
- F. Place endotracheal tube (ETT) into right corner of mouth and pass through the vocal cords; stop just after the cuff disappears behind vocal cords. Do not attempt blind placement of ET tube if the cords are not visualized. Application of cricoid pressure by an assistant may aid in visualization of cords. If intubation is unsuccessful after 30 seconds, stop and resume bag and mask ventilation before reattempting. If necessary, use stilette to maintain the shape of the ETT (a hockey stick shape may be helpful); remove stilette after intubation.
- G. Inflate cuff with syringe keeping cuff pressure ≤ 20 cm H₂O and attach the tube to an Ambu bag or ventilator. Confirm bilateral, equal expansion of chest and equal bilateral breath sounds. Auscultate abdomen to confirm that the ETT is not in esophagus. A rapid rise in pulse oximetry, or a rise in end tidal CO₂ confirms proper placement if available. If there is any question about proper ETT location, repeat laryngoscopy with tube in place to be sure it is endotracheal; remove

tube immediately if there is any doubt about proper location. Secure the tube with tape and note centimeter mark at the mouth. Suction the oropharynx and trachea.

- H. Confirm placement with a chest X-ray (tip of ETT should be between the carina and thoracic inlet, level with of the top of the aortic notch).

II. Nasotracheal Intubation

- A. Nasotracheal intubation is preferred if prolonged intubation is anticipated (increased patient comfort) or if extensive oral trauma. Patient must be spontaneously breathing. Nasotracheal intubation is much more difficult than orotracheal intubation and should not be attempted in emergent situations.

1. Spray Nasal Passage with Vasoconstrictor: Phenylephrine 0.25% (Neo-Synephrine). If sedation is required before nasotracheal intubation, administer fentanyl (Sublimaze) 25 to 50 mcg increments with midazolam hydrochloride (Versed) 1-2 mg increments.

2. Lubricate nasal airway with lidocaine ointment.

3. Tube Size

Women 7.0 mm tube

Men 8.0 mm tube

4. **Place the nasotracheal tube** into nasal passage and guide it into nasopharynx along a U-shaped path.
5. Monitor breath sounds by listening and feeling the end of tube. As the tube enters the oropharynx, gradually guide the tube downward. Listen at the end of tube until breath sounds are loudest, then advance tube through cords during inspiration.
6. If the sounds stop, withdraw the tube 1-2 cm until breath sounds are heard again. Reposition the tube, and, if necessary, extend the head and advance.
7. If difficulty is encountered, perform direct laryngoscopy and insert under direct visualization.
8. Successful intubation occurs when the tube passes through the cords; a cough may occur and breath sounds will reach maximum intensity if the tube is correctly positioned. Confirm correct placement by checking for bilateral breath sounds and expansion of chest.
9. Confirm placement with chest x-ray.

Ventilator Management

- A. **Indications for Ventilatory Support:** Respiratory rate >35 , vital capacity <15 mL/kg, negative inspiratory force ≤ -25 cm H₂O, pO₂ <60 mm Hg on 50% oxygen, pH < 7.2 , pCO₂ ≥ 55 ; severe, progressive, symptomatic hypercapnia or hypoxia.

- B. **Initial Orders:** FiO₂ = 100%, PEEP = 5-10 cm H₂O, assist control 8-14 breaths/min, tidal volume = 800 mL (10-15 mL/kg ideal body weight), set rate to provide a minute ventilation (VE) of approximately 10 L/min. Alternatively, use intermittent mandatory ventilation mode with tidal volume and rate set to achieve near-total ventilatory support. Consider pressure support at 5-15 cm H₂O.

- C. **If Decreased Minute Ventilation:** Evaluate patient and rule out complications (endotracheal tube malposition, secretions, bronchosp-

asms, pneumothorax, worsening pulmonary disease). Readjust ventilator rate to maintain mechanically assisted minute ventilation of 10 L/min. If peak AWP is > 45 cm H₂O, consider decreasing tidal volume to 7-8 mL/kg or decreasing ventilator flow rate, or change to pressure control mode.

- D. ABG should be measured in 30 min**, CXR for tube placement, pulse oximeter, arterial line, and/or monitor end tidal CO₂. Maintain oxygen saturation $> 90 - 95\%$.
- E. If Arterial Saturation $\geq 98\%$ and $pO_2 > 100$** , reduce FIO₂ (each 1% decrease in FIO₂ reduces pO₂ by up to 7 mm Hg); once FIO₂ is less than 50%, may reduce PEEP by increments of 2 cm H₂O. Maintain O₂ saturation of greater than 90% (pO₂ > 60). If known coronary artery disease, or if septic patient with flow limited oxygen consumption, attempt to maintain pAO₂ $> 95\%$.
- F. If Arterial Saturation $< 90\%$ and $pO_2 < 60$** , increase FIO₂ up to 60-100%, then increase PEEP by increments of 3-5 cm H₂O (PEEP > 15 mmHg requires PA catheter), or switch to pressure control mode, especially if poor compliance or high pressure. Add additional PEEP until oxygenation is adequate with an FIO₂ of $< 60\%$. High levels of PEEP may be contraindicated in patients with elevated intracranial pressure. PEEP may have deleterious effects on cardiac filling and hence cardiac output, especially if filling pressures are low.
- G. If pH is Excessively Low**, (pH < 7.33 due to respiratory acidosis/hypercapnia) increase rate and/or tidal volume. Keep peak airway pressure $< 40-50$ cm H₂O.
- H. If pH is High**, (> 7.48 due to respiratory alkalosis/hypocapnia) reduce rate and/or tidal volume. If patient is breathing rapidly above ventilator rate, change to IMV from assist control, or sedate patient.
- I. If Patient is "Fighting" Ventilator**, consider assist control or synchronized intermittent mandatory ventilation mode, or add sedation with or without paralysis. Never use paralytic agents without concurrent analgesia/sedation. Exclude complications or other causes of agitation.
- J. Sedation (if pH > 7.55)**
 - 1. Morphine Sulfate 2-5 mg IV q5min, max dose 20-30 mg OR Infusion 0.03-0.05 mg/kg/h continuous IV infusion (50-100 mg in 500 mL D5W) titrated **OR**
 - 2. Propofol (Diprivan) 2.0-2.5 induction 2.0-2.5 mg/kg IV (40 mg q10 sec) maintenance: 100-200 mcg/kg/min in D5W.
 - 3. Diazepam (Valium) 2-5 mg slow IV q2h prn agitation, max dose 20-30 mg **OR**
 - 4. Lorazepam (Ativan) 1-2 mg IV q1-2h prn sedation **OR**
 - 5. Midazolam (Versed) 1-2 mg IV boluses until sedated **OR**
 - 6. Beware of cardiac depressant effects of benzodiazepines if patient has marginal cardiac status.
- K. Paralysis (with simultaneous sedation)**
 - 1. Succinylcholine (Anectine; very short acting) 0.6-1.0 mg/kg **OR**
 - 2. Atracurium (Tracrium; short acting) 0.5 mg/kg IV, then 0.3-0.6 mg/kg/h infusion. **OR**
 - 3. Vecuronium (Norcuron; intermediate acting) 0.1 mg/kg IV, then 0.06 mg/kg/h IV infusion or q1h prn **OR**
 - 4. Pancuronium bromide (Pavulon; long-acting) 0.08 mg/kg IV, then 0.03 mg/kg/h infusion or q1h IV prn (adjust if renal dysfunction is

present).

Note: Follow level of paralysis with nerve stimulation.

- L. **Loss of Tidal Volume:** Assess breath sounds and rule out pneumothorax. Check for a leak in the ventilator or inspiratory line. Check for a poor seal between the endotracheal tube cuff or malposition of the cuff in the subglottic area. If a chest tube is present, check for airleak.

Acute Epistaxis

Almost all persons have experienced a nosebleed at some time, and most nosebleeds resolve without requiring medical attention. Prolonged epistaxis, however, can be life-threatening, especially the elderly or debilitated.

I. Pathophysiology

- A. **Anterior epistaxis**, in the anterior two thirds of the nose, is visible on the septum, and is the most common type of epistaxis. Anterior bleeding can often be resolved by pinching the cartilaginous part of the nose.
- B. **Posterior epistaxis** from the posterior third of the nose accounts for 10% of nosebleeds. Bleeding is profuse because of the larger vessels in that location. Posterior epistaxis usually occurs in older patients, who have fragile vessels because of hypertension, atherosclerosis, coagulopathies, or weakened tissue. Posterior bleeds require aggressive treatment and hospitalization.
- C. The anterior portion of the septum has a rich vascular supply, known as Kiesselbach's plexus, or Little's area, and most epistaxis originates in this region. Posterior hemorrhages originate from larger vessels near the sphenopalatine artery, behind the middle turbinate.

II. Causes of Epistaxis

- A. **Trauma:** Nose picking, nose blowing, or sneezing can tear or abrade the mucosa and cause bleeding. Other forms of trauma include nasal fracture and nasogastric and nasotracheal intubation.
- B. **Desiccation:** Cold, dry air, and dry heat contribute to an increased incidence of epistaxis during the winter.
- C. **Other causes of nasal drying** include dehydration (eg, from poorly controlled diabetes mellitus), nasal sprays (eg, corticosteroids and cromolyn), and nasal oxygen therapy.
- D. **Irritation:** Upper respiratory infections, sinusitis, allergies, topical decongestants, and cocaine sniffing cause increased vulnerability to bleeding.
- E. **Hereditary Hemorrhagic Telangiectasia:** This autosomal dominant condition weakens the capillaries and causes bleeding.
- F. **Nasal Septal Disease:** Less common causes of anterior epistaxis include Wegener's granulomatosis, mid-line destructive disease, tuberculosis, and syphilis.
- G. **Systemic Disease:** Epistaxis will be exacerbated by coagulopathy, blood dyscrasia, thrombocytopenia, or anticoagulant medication (NSAIDs, warfarin), hepatic cirrhosis, and renal failure.
- H. **Hypertension** complicates active bleeding by promoting rigid arteries,

and arteriosclerosis weakens vessels and inhibits vasoconstriction. These risk factors contribute to the posterior epistaxis most often seen in elderly patients.

- I. **Tumors:** Juvenile nasopharyngeal angiofibroma, which occurs most frequently in teenage boys, is benign. It can cause profuse, life-threatening posterior epistaxis. Inverted papilloma less commonly presents with bleeding. Malignant neoplasms such as squamous cell carcinoma, adenocarcinoma, melanoma, esthesioneuroblastoma, and lymphoma can cause epistaxis.
- J. **Foreign Bodies:** A foreign body should be considered when a child has unilateral nasal obstruction, new-onset snoring, halitosis, or epistaxis.

III. Clinical Evaluation of Epistaxis

- A. Hemodynamic evaluation for tachycardia, hypotension, lightheadedness should be completed immediately. Hypovolemic patients should be resuscitated with fluids and packed red blood cells.
- B. After stabilization, the site, cause, and amount of bleeding is determined. Most patients require no immediate resuscitation. Posterior epistaxis in an elderly and debilitated patient can be life-threatening because of aspiration, hypoxia, exsanguination, or myocardial infarction.
- C. **Determine the side on which the bleeding occurred:** Unilateral nosebleeding suggests anterior epistaxis in Kiesselbach's plexus. Bilateral bleeding suggests posterior epistaxis caused by overflow around the posterior septum.
- D. **Determine whether epistaxis is anterior or posterior:** When the patient is upright, blood drains primarily from the anterior part of the nose in anterior bleeding, or it drains from the nasopharynx in posterior bleeding.
- E. **Assess the duration** of the nosebleed and any inciting incident (eg, trauma). Swallowed blood from epistaxis may cause melena.
- F. **Medical History:** Hypertension, bleeding disorders, diabetes, alcoholism, liver disease, pulmonary disease, cardiac disease, arteriosclerosis should be assessed.
- G. **Medications** including aspirin or aspirin-containing products, other NSAIDs, warfarin, nasal sprays, and oxygen via nasal cannula should be sought.
- H. **Blood Tests**
 - 1. Hematologic tests include CBC, platelet counts, prothrombin time, and partial thromboplastin time, and blood for type and cross in case a transfusion is needed.
 - 2. The hematocrit does not immediately drop in acute hemorrhage because time is required for fluid dilution of blood.

IV. Location of the Site of Bleeding

- A. **Sedation:** When sedation is required, midazolam (Versed), 1-2 mg IV in adults and 0.035-0.2 mg/kg IV in children is recommended; overmedication may threaten the cough reflex that protects the airway.
- B. **Drape the patient**, and furnish a spit basin. Keep the patient sitting upright or leaning forward. Wear a gown, gloves, mask, and protective eyewear because patients may inadvertently cough blood.
- C. **A nasal speculum** and a suction apparatus with a #10 Frazier tip are

used to aspirate blood from the nose and oropharynx.

D. Anesthesia and Vasoconstriction: A cotton-tipped applicator or cotton pledget is used to apply a topical anesthetic (eg, 1% tetracaine or 4% lidocaine) and a topical vasoconstrictor (eg, 1% ephedrine, 1% phenylephrine, or 0.05% oxymetazoline) to the entire nasal mucosa, especially to the septum, inferior turbinate, posterior middle turbinate, and superior mucosa. These agents take effect in 5 to 10 minutes. If a bleeding site is observed, press the vasoconstrictor applicator directly to that site.

E. Visualization of Bleeding Site

1. Use the nasal speculum to facilitate exposure, taking care to minimize further mucosal injury. Apply suction to remove blood and clots.
2. Localize active bleeding. Kiesselbach's plexus and the inferior turbinate are the most frequent sites of bleeding.
3. If blood is spilling from the superior aspect of the nose, the ethmoidal arteries may be involved, usually following facial trauma.
4. Posterior bleeding may be located by applying suction; when the suction tip is at the site of bleeding, blood will no longer well up.

V. Management of Hemorrhage

A. Anterior septal hemorrhage can sometimes be stopped by nose pinching alone. Have the patient sit upright to decrease venous pressure. If bleeding continues, application of the topical anesthetic and vasoconstrictor may stop it.

B. Cauterization

1. Bleeding sites that can be visualized should be cauterized with silver nitrate sticks; they work best in a dry field accomplished by vasoconstriction and suction.
2. Press or roll the silver nitrate over the bleeding site for several seconds, allowing a gray eschar to form.
3. The septum should not be cauterized bilaterally at the same level because it may cause cartilaginous necrosis and septal perforation.

C. Hemostatic Agents

1. Application of a hemostatic material (eg, microfibrillar collagen or oxidized regenerated cellulose) to the bleeding site may be useful. These products do not require removal because they dissolve in the nose after several days. The patient should lubricate the nose regularly with saline nasal spray and to use bacitracin ointment tid.

D. Packing: Gauze packing or a sponge pack applied to the anterior portion of the nose are used as a tamponade for uncontrolled bleeding sites. These packs stay in place for 5 days.

E. Anterior Nasal Pack

1. The pack consists of 72 inches of half-inch gauze impregnated with antibiotic ointment (eg, bacitracin). Topical anesthesia is necessary.
2. Use the nasal speculum to open the vestibule vertically. With the bayonet forceps, grasp the gauze approximately 10 cm from the end; place a layer along the floor of the nose all the way back to the nasopharynx. The loose end should protrude from the nose. Reposition the speculum on top of the first layer and apply another

layer. Fill the entire nose with gauze, and apply a nasal drip pad.

3. **An Oral Broad-spectrum Antibiotic**, such as cephalexin (Keflex), 500 mg orally every 6 hours, is given while the pack is in place to prevent secondary sinusitis or toxic shock syndrome.
4. The pack may obstruct the nasolacrimal duct, causing tearing or retrograde bleeding from the lacrimal puncture at the medial canthus.
5. The pack is removed after 5 days.

F. Sponge Pack

1. A newer variation of the anterior pack consists of a dry, compressed sponge. The sponge is lubricated with an antibiotic ointment, then placed into the nasal cavity. Once moistened with blood or saline, the sponge expands, filling the nasal cavity and exerting gentle pressure on the bleeding site.
2. Moisten the pack before removal after 5 days.

G. Posterior Pack

1. Posterior bleeding requires a posterior a pack, and the patient should be admitted to the hospital.
2. The posterior pack requires a sphenopalatine block and topical anesthetic.
3. The posterior pack is made by sewing together two tonsil tampons with 0-silk, leaving two 8-inch tails, and lubricating the tampons with antibiotic ointment.
4. Pass the red rubber catheter through the involved nostril to the oropharynx and out the mouth. Attach the free ends of the silk sutures to the oral end of the catheter, and pull the catheter back out of the nose, pulling the tampons into the nasopharynx while guiding them with your fingers, and lodge them snugly into the involved choana. Be sure the uvula is free.
5. Trim the loose cotton tampon tails at the level of the uvula to facilitate pack removal.
6. Have an assistant hold the packs firmly in place by applying traction to the silk tails as they exit the nostril.
7. Place a tight anterior gauze pack, and tie the tails around one or two dental rolls to stabilize the tampons.
8. The patient is admitted to the hospital for airway observation, administration of humidified air or oxygen, mild sedation, and antibiotics. Elevate the head of the bed at least 30 degrees. Cold foods and liquids are given, and ambulation is allowed after 12-24 hours.
9. The pack is removed after 5 days.

H. Balloon Pack

1. A popular alternative to the tampon posterior pack is a 14-French, 30-mL balloon Foley catheter.
2. Use the same anesthetic as for the tampon posterior pack, and cover the balloon with antibiotic ointment, and insert it through the nostril until the tip can be seen in the nasopharynx.
3. Inflate the balloon with 10 to 12 mL of saline or water, and lodge it in the choana using anterior traction. Do not inflate the balloon with air, because it will slowly leak out.
4. Place an anterior pack around the catheter back to the balloon.
5. Secure the Foley catheter under traction, using an umbilical

clamp, protecting the ala with thick gauze pads.

6. The advantages of this procedure include ease of placement, relative patient comfort, and the ability to test adequate hemostasis by deflating the balloon. Reinflate it if bleeding recurs.

VI. Management of Refractory Epistaxis: Patients who continue to bleed after a snug posterior pack is in place, may require endoscopic cautery, radiographic embolization or surgical ligation of the arterial supply to the bleeding area.

VII. Prevention of Nosebleeds

- A. Patients should avoid nose picking or excessive nose blowing.
- B. The fingernails of children should be trimmed regularly.
- C. A bedside humidifier should be used at night, especially during the winter.
- D. Nasal saline spray is used frequently to soften and loosen crusts.
- E. Drink fluids liberally to stay well hydrated.
- F. Antibiotic ointment or petroleum jelly is applied to nasal vestibules two or three times daily to minimize crusting.
- G. Keep hypertension under control.
- H. Avoid prolonged NSAID use.
- I. Avoid rigorous physical activity for 7 days after serious epistaxis.

Disorders of the Alimentary Tract

Acute Abdomen

I. Clinical Evaluation

- A. Duration, acuity, and progression of pain; exact location of maximal pain at onset and at present; diffuse or localized. Time course of pain (constant, intermittent, decreasing, increasing).
- B. Character at onset and at present (crampy, sharp, dull); constant or intermittent; radiation (to shoulder, mid-back, groin); sudden or gradual onset.
- C. Effect of eating, vomiting, defecation, passing of gas, urination, inspiration, movement, position, last bowel movement, menstrual period. Similar episodes in past.
- D. **Associated Symptoms:** Fever, chills, nausea, vomiting (bilious, feculent, food, blood, coffee grounds); vomiting before or after onset of pain; chest pain; constipation, change in bowel habits or stool caliber; obstipation (inability to pass gas); diarrhea, hematochezia (rectal bleeding), melena (black stools), dysuria, anorexia, weight loss, early satiety.
- E. **Past Medical History:** Abdominal surgery (appendectomy, cholecystectomy), hernias, gallstones; coronary disease, kidney stones; alcohol intake, dyspepsia. Past treatment or testing, endoscopies, x-rays.
- F. **Aggravating or Relieving Factors:** Fatty food intolerance, medications, aspirin, NSAID's. Use of narcotics, anticholinergics, laxatives, antacids.

II. Physical Exam

- A. **General:** Degree of distress; severity of pain; nutritional status; activity (moving to relieve pain or lying still to prevent pain).
- B. **Vital Signs:** T (fever), P (tachycardia), BP (hypotension), R (tachypnea).
- C. **HEENT:** Oral signs of dehydration (lack of oral moisture, fissured tongue); signs of upper respiratory infection.
- D. **Chest:** Rhonchi or rales may indicate lower lobe pneumonia that may cause upper abdominal pain.
- E. **Abdomen:**
 - 1. **Inspection:** Scars, evidence of trauma, visible peristalsis (small bowel obstruction), distention.
 - 2. **Auscultation:** Absent bowel sounds (paralytic ileus or late obstruction), high-pitched rushes (obstruction), bruits (ischemic colitis).
 - 3. **Palpation:** Begin palpation in quadrant diagonally opposite to point of maximal pain with patient's legs relaxed and slightly flexed. Bimanual palpation of flank may reveal renal disease or retrocecal appendicitis. Rebound tenderness, hepatomegaly,

splenomegaly, masses; hernias (incisional, inguinal, femoral). Pulsating masses; costovertebral angle tenderness. Shifting dullness (ascites). Cutaneous hyperesthesia (pain with light touch, indicates parietal peritoneal inflammation of appendicitis).

4. **Specific Abdominal Signs**

- a. **Murphy's sign:** Inspiratory arrest with right upper quadrant palpation, cholecystitis.
- b. **Charcot's Triad:** Right upper quadrant pain, jaundice, fever; gallstones.
- c. **Courvoisier's sign:** Palpable, nontender gallbladder with jaundice, pancreatic malignancy.
- d. **McBurney's Point Tenderness:** Located two thirds of the way between umbilicus and anterior superior iliac spine, appendicitis.
- e. **Iliopsoas Sign:** Elevation of legs against examiner's hand causes pain, retrocecal appendicitis.
- f. **Obturator Sign:** Flexion of right thigh and external rotation of thigh causes pain in pelvic appendicitis.
- g. **Rovsing's Sign:** Manual pressure and release at left lower quadrant colon causes referred pain at McBurney's point (referred rebound tenderness from appendicitis).
- h. **Cullen's Sign:** Bluish periumbilical discoloration, peritoneal hemorrhage.

5. **Percussion:** Loss of liver dullness indicates perforated viscus and free air in peritoneum; liver and spleen span by percussion.

F. **Rectal:** Masses, tenderness, feces; gross or occult blood. Bimanual palpation of the lower abdomen may elicit concealed tenderness or mass.

G. **Genital/Pelvic Examination:** Adnexal tenderness, cervical discharge, uterine size, masses, cervical motion tenderness.

H. **Extremities:** Femoral pulses, popliteal pulses (absent pulses may indicate ischemic colitis).

I. **Skin:** Jaundice, dependent purpura (mesenteric infarction), petechia (gonococcemia).

J. **Stigmata of Liver Disease:** Spider angiomas, periumbilical collateral veins (Caput medusae), gynecomastia, ascites, hepatosplenomegaly, testicular atrophy.

III. **Laboratory Evaluation of the Acute Abdomen**

A. CBC, electrolytes, liver function tests, amylase, lipase, UA. Serum beta-HCG, ECG; chest x-ray: free air under diaphragm.

B. **Acute Abdomen Series:** Includes upright PA chest, plain abdominal film ("flat plate"), upright film and left lateral decubitus; check for flank stripe, air fluid levels, mass effect, calcifications, fecaliths, absent bowel gas in RLQ (appendicitis), aortic aneurysm. Free air, widening of spaces between loops of bowel (thickening of bowel wall), and "ground glass" appearance of ascites may be seen.

IV. **Differential Diagnosis**

A. **Generalized Pain:** Generalized peritonitis, intestinal infarction, obstruction, diabetic ketoacidosis, sickle crisis, acute porphyria, penetrating posterior duodenal ulcer, psychogenic.

- B. Epigastrium:** Gastritis, peptic ulcer, esophagitis, gastroenteritis, pancreatitis, perforated viscus, intestinal obstruction, myocardial infarction, aortic aneurysm, ileus.
- C. Right Upper Quadrant:** Biliary colic, cholecystitis, cholangitis, hepatitis, gastritis, pancreatitis, pneumonia, peptic ulcer, gastroesophageal reflux disease, retrocecal appendicitis, hepatic metastases, gonococcal perihepatitis (Fitz-Hugh-Curtis syndrome).
- D. Left Upper Quadrant:** Peptic ulcer, gastritis, esophagitis, gastroesophageal reflux, pancreatitis, pericarditis, myocardial ischemia, pneumonia, splenic rupture, pulmonary embolus.
- E. Left Lower Quadrant:** Diverticulitis, intestinal obstruction, colitis, strangulated hernia, inflammatory bowel disease, pyelonephritis, nephrolithiasis, mesenteric lymphadenitis, mesenteric ischemia or thrombosis, aortic aneurysm rupture, volvulus, intussusception, salpingitis, ovarian cyst, ectopic, testicular torsion, psychogenic.
- F. Right Lower Quadrant:** Appendicitis, diverticulitis, salpingitis, renal calculus, intussusception, ruptured ectopic pregnancy, hemorrhage or rupture of ovarian cyst, perforated cecal diverticulum, regional enteritis.
- G. Hypogastric/Pelvic:** Cystitis, salpingitis, ectopic, endometriosis, endometritis, appendicitis, diverticulitis, ovarian cyst torsion; distended bladder, hernia, nephrolithiasis, prostatitis, carcinoma.

Management of the Acute Abdomen

I. Acute Management

- A. Evaluation** should be timely with a decision made on operation, or non-operation and observation. Admit for repeat examination even if the need for operation remains uncertain.
- B. Analgesics and antibiotics** should be considered only after a presumptive diagnosis has been made. Narcotics and sedatives should be withheld until a diagnosis has been determined.
- C. Patient should be NPO** until a definite diagnosis is determined and treatment is initiated.
- D. IV fluids:** Maintenance fluids plus expected fluid losses. Replace prior loss if vomiting or dehydration. Nasogastric intubation and suction should be initiated if the patient is vomiting, bleeding, or has signs of intestinal obstruction.
- E. Vital signs** q1hour. Place Foley catheter and monitor urine output.
- F. Serial abdominal examinations** should be performed until definitive diagnosis has been made or the patient improves. Draw serial CBC q4-6 hours. If necessary, give maintenance medications by IV, IM, or by suppository.
- G.** The patient should be considered to have appendicitis or other serious abdominal pathology until determined otherwise.

II. Laboratory Testing

- A.** CBC with differential, urinalysis, electrolytes, abdominal and chest x-rays. Culture of cervical discharge.
- B.** Ultrasound of right upper quadrant (and upper abdomen if gallstones, common bile duct obstruction, or pancreatitis suspected). Ultrasound

of lower abdomen and pelvis if symptoms of diverticulitis, pelvic inflammatory disease, appendicitis.

- C. Low pressure water soluble contrast enema or CT with contrast per rectum if suspect diverticulitis.
- D. Intravenous pyelogram (IVP) if gallstones suspected.

Appendicitis

I. Clinical Evaluation

- A. **History:** Abdominal pain beginning in the epigastrium or mid abdomen then migrating to the right lower quadrant within 6 hours. Alteration in bowel function (diarrhea or constipation). Anorexia, nausea; vomiting after the onset of pain. Perforation may cause generalized peritonitis that is manifest by pain that changes from localized to diffuse.
- B. **Physical**
 - 1. Elevated temperature; tenderness at McBurney's point (2/3 of the way between the umbilicus and anterior superior iliac spine), decreased bowel sounds.
 - 2. Rebound tenderness, rigidity. Palpation of the left lower quadrant often causes referred pain to the right lower quadrant (Rovsing's sign). A mass may indicate an abscess.
 - 3. Iliopsoas sign (elevation and flexion-extension of right leg elicits back pain).
 - 4. Obturator sign (flexion of knee and rotation of leg medially causes worsening of pain, pelvic appendix).
 - 5. Rectal or pelvic exam elicits pain on right side; cutaneous hyperesthesia (hypersensitivity to touch in right lower quadrant).

II. Laboratory Evaluation

- A. WBC count (leukocytosis and left shift). Urinalysis may reveal protein, WBC's or RBC's.
- B. Upright abdominal x-ray: Absent bowel gas in the right lower quadrant with normal gas pattern in the remainder of the abdomen. Loss of the right flank stripe; a fecalith may sometimes be visible.
- C. Ultrasonography may reveal swollen appendix.
- D. Laparoscopy may be indicated if a gynecologic diagnoses is suspected.

III. Differential Diagnosis: Appendicitis, PID, intestinal obstruction, strangulated hernia, inflammatory bowel disease, gastroenteritis, pyelonephritis, mesenteric adenitis, mesenteric ischemia or thrombosis; nephrolithiasis, volvulus, intussusception, sickle crisis, ovarian cyst, ectopic, salpingitis, carcinoma, ketoacidosis, zoster neuritis, spinal arthritis, testicular torsion, psychogenic.

IV. Treatment

- A. If a clinical suspicion of appendicitis exist, then early appendectomy via right lower quadrant incision should be performed.
- B. A diagnosis should be determined within 24 hours of admission, and a decision should be made to operate or to provide conservative

therapy.

- C. If a periappendiceal phlegmon is present then selected patients may be treated conservatively with broad spectrum IV antibiotics, and no oral intake. Antibiotics should cover gram negative and anaerobic organisms. Interval appendectomy can be done in 6 weeks.
- D. For an appendiceal abscess, give IV antibiotics, drain abscess via RLQ incision and remove appendix.
- E. Antibiotic (Cefoxitin, Cefotan, Timentin, or Zosyn) is given IV until resolution of ileus, absence of tenderness, and afebrile for 48 hours; usually within 5 days.

Appendectomy Surgical Technique

1. After induction of anesthesia, place an incision over any obvious appendiceal mass if palpable. If no mass is present, make a transverse skin incision over McBurney's point, located two thirds of the way between the umbilicus and anterior superior iliac spine.
2. Incise the subcutaneous tissues in the line of the transverse incision, and incise the external oblique aponeurosis in the direction of its muscle fibers. Spread the muscle with a Peon hemostat. Incise the internal oblique fascia and spread the incision in the direction of its fibers. Sharply incise the transversus abdominis muscle, transversalis fascia, and peritoneum. Note the presence and nature of peritoneal fluid, and send purulent fluid for Gram's stain and aerobic and anaerobic culture.
3. Identify the base of the cecum by the converging taenia coli, and raise the cecum, exposing the base of the appendix. Hook an index finger around the appendix, and gently break down any adhesions to adjacent tissues. Use gauze packing to isolate the inflamed appendix, and stabilize the appendix with a Babcock forceps.
4. Divide the mesoappendix between clamps, then firmly ligate below the clamps with 000 silk or polyglycolic acid sutures. Apply an encircling purse-string suture of 000 silk at the end of the cecum about 0.8 cm from the base of the appendix. Place a hemostat at the proximal base of the appendix, and crush the appendix. Remove the hemostat and reapply it distal to the crush, on the appendix. Use an 0 chromic catgut suture to ligate the crushed area below the hemostat.
5. Transect the appendix against the clamp. Invert the stump into the cecum with the purse string, and tie the purse-string suture, burying the stump. Irrigate the peritoneum with normal saline, and examine the mesoappendix and abdominal wall for hemostasis, then close the peritoneum with continuous 000 catgut suture.
6. Close the internal oblique and transversus abdominis with interrupted 0 chromic catgut. Close the external oblique as a separate layer. Close the skin and subcutaneous tissues.
7. Drains should be placed if perforation has occurred and a well-defined residual abscess cavity is present. Use a soft rubber Penrose drain and bring out through a separate stab incision in the lateral abdominal wall, located as dependently as possible.

F. Intravenous Antibiotics

1. Antibiotic prophylaxis should include coverage for bowel flora,

including aerobes and anaerobes.

2. Recommended prophylaxis: Cefoxitin (Mefoxin) or cefotetan (Cefotan) or piperacillin/tazobactam (Zosyn) should be given prophylactically before the operation and discontinued after one or two doses postoperatively unless the appendix has perforated.
3. If perforation is found, IV antibiotics should be continued for approximately 5-10 days. Check culture on third postoperative day and change antimicrobials if a resistant organism is present.

Abdominal Hernias

I. Classification of Hernias

- A. A hernia is an abnormal opening in the abdominal wall, with or without protrusion of an intra-abdominal structure. The most common groin hernia in either males or female is the indirect inguinal hernia. Femoral hernias are more common in females than in males.
- B. **Incarceration:** A hernia that can not be reduced; may be acute or chronic; painful or asymptomatic.
- C. **Strangulated Hernia:** An incarcerated hernia with compromised blood supply.
- D. **Sliding Hernia:** A hernia in which a portion of the hernia wall consists of either cecum or sigmoid.
- E. **Richter's Hernia:** Only a "knuckle" of bowel enters the hernia sac, and only one wall of the bowel is within the sac. Bowel incarceration can occur without bowel obstruction.

II. Anatomy of the Inguinal Area

- A. **Layers of Abdominal Wall:** Skin, subcutaneous fat, Scarpa's fascia, external oblique, internal oblique, transversus abdominus, transversalis fascia, peritoneum.
- B. **Hesselbach's Triangle:** A triangle formed by the lateral edge of rectus sheath, the inferior epigastric vessels, and the inguinal ligament (direct hernias occur here).
- C. **Inguinal Ligament:** Ligament running from anterior superior iliac spine to the pubic tubercle.
- D. **Lacunar Ligament:** Reflection of inguinal ligament from the pubic tubercle onto the iliopectineal line of the pubic ramus.
- E. **Cooper's Ligament:** Strong, fibrous band located on the iliopectineal line of superior pubic ramus (used for McVay repair).
- F. **External Inguinal Ring:** Opening in the external oblique aponeurosis; the ring contains the ilioinguinal nerve and spermatic cord or round ligament.
- G. **Internal Ring:** Bordered superiorly by internal oblique muscle and inferomedially by the inferior epigastric vessels and the transversalis fascia.
- H. **Processus Vaginalis:** A diverticulum of peritoneum which descends with testicle, and lies adjacent to the spermatic cord. May enlarge to become the sac of an indirect inguinal hernia.
- I. **Femoral Canal:** Formed by the borders of the inguinal ligament, lacunar ligament, Cooper's ligament, and femoral sheath.

III. Classification of Hernias

- A. Indirect Inguinal Hernia:** Hernia sac is located anteromedial to the spermatic cord; the sac exits through the internal ring and lies lateral to the inferior epigastric artery. The indirect inguinal hernia is the most common inguinal hernia in either sex.
- B. Direct Inguinal Hernia:** Hernia passes through Hasselbalch's triangle and lies medial to inferior epigastric artery.
- C. Pantaloon Hernia:** Combination of both direct and indirect inguinal hernias.
- D. Femoral Hernia:** Hernia passes through the femoral canal and lies medial to the femoral vein.
- E. Umbilical Hernia:** Hernia passes through the umbilical ring. This hernia is usually congenital, but may be acquired, especially in patients with ascites.
- F. Incisional Hernia:** Hernia appears after surgery through a previous fascial closure.
- G. Epigastric Hernia:** Caused by a defect in the linea alba, and located above umbilicus; frequently multiple.
- H. Obturator Hernia:** Hernia passes through obturator foramen.
- I. Spigelian Hernia:** Hernia occurring at semilunar line, at the lateral edge of the rectus muscle.
- J. Petit's Hernia:** Hernia passes through lumbar triangle.
- K. Perineal Hernia:** Hernia passes through a defect in the muscular pelvic floor; rectal prolapse often occurs.
- L. Sciatic Hernia:** Hernia sac passes through greater or lesser sacrosciatic foramen; rarest type of hernias.

Evaluation of the Hernia Patient

I. Clinical Evaluation

- A. History:** Patient complains of a soft mass that increases in size with straining. The mass may reduce manually or spontaneously. Pain may occur with straining. History of symptoms of small bowel obstruction may be present.
- B.** Hernias may be caused by obesity, chronic cough, straining at stools due to constipation; straining at urination; ascites, previous abdominal surgery.
- C. Physical Exam:** Palpable mass which increases in size when the patient strains; examine in the upright and supine positions; examine with patient relaxed and straining.
- D. Examination for Hernias:** The examiner places index finger upward into scrotum, and into the external inguinal ring while patient strains or coughs. Direct hernias are felt on the medial side of the examiner's finger; indirect hernias are felt on the tip of the finger.
- E.** Examine for prostatic enlargement and complete a rectal exam. Consider flexible sigmoidoscopy/colonoscopy.

Surgical Repair Techniques for Hernias

I. Surgical Techniques

- A. Bassini Repair:** Approximation of conjoint tendon and the transversalis fascia to the edge of inguinal ligament.
- B. McVay Repair (Cooper's Ligament Repair):** Approximation of conjoint tendon to Cooper's ligament; a relaxing incision in rectus sheath may be added.
- C. Halsted Repair:** Same as Bassini repair, except imbricated external oblique is used to reinforce the repair beneath the spermatic cord.
- D. Ligation of Sac:** Useful only in children who have only a simple patency of processus vaginalis.
- E. Pre-Peritoneal Hernia Repair:** Hernia defect is exposed and repaired from beneath fascia in preperitoneal space.
- F. Femoral Hernia Repair:** Use McVay or Cooper's ligament repair to repair femoral hernias. Do not attempt reduction of tender femoral hernias.
- G. Ventral Hernias:** Primary repair of fascial defect. Usually requires marlex mesh.

II. Reduction of Incarcerated Hernias

- A.** Place patient in the Trendelenburg's position and sedate.
- B.** Apply gentle continuous compression. No reduction should be attempted if possible strangulation. Strangulation is indicated by severe tenderness, induration, erythema, leukocytosis.
- C.** Strangulated hernias should be treated in operating room. Do not attempt to reduce incarcerated femoral hernias.

Hernia Repair Surgical Technique

I. Indirect Hernia

1. Prep and draped the skin of the abdomen, inguinal region, upper thigh, and external genitalia. Place the incision 1 cm above and parallel to the inguinal ligament. Begin the incision at a point just above, and medial to, the pubic tubercle, and extend it to a point two-thirds the distance to the anterior iliac spine. Incise the subcutaneous fat in the length of the incision down to the external oblique aponeurosis. Clear the external oblique muscle of overlying fat and identify the external inguinal ring. Incise the aponeurosis of the external oblique, beginning laterally and splitting the aponeurosis in the direction of its fibers, taking care not to injure the underlying ilioinguinal nerve. Expose the inguinal canal in which the spermatic cord and the indirect inguinal hernia are located.
2. Use blunt dissection to mobilize the spermatic cord and the associated hernia up to the level of the pubic tubercle. Lift these structures up from the floor of the inguinal canal, and encircle with a Penrose drain. Retract the drain anteriorly, and free the rest of the cord from the floor of the canal. Use sharp and blunt dissection to incise the anterior muscular and fascial investments of the cord.
3. Sharply incise the internal spermatic fascia, and locate the hernial sac; identify the spermatic artery, venous plexus, and vas deferens

before opening the sac.

4. Incise the indirect hernial sac, anteriorly along its long axis, using caution if the sac is filled with small bowel or omentum. Place an index finger inside the hernial sac, and separate the sac from surrounding cord structures using sharp and blunt dissection. Carry dissection proximally to the internal inguinal ring.
5. Close the sac with a circumferential purse-string suture on an atraumatic, gastrointestinal needle. Tie this suture, being careful that no abdominal organs are within the purse string (high ligation of the sac). Reinforce this ligation by placing another transfixion suture through the sac, 1 mm distal to the purse-string suture. Transect the sac a few millimeters below the second suture, and cut both sutures and allow sac to retract into the retroperitoneum. Remove the sac. Inspect the floor of the inguinal canal, and if there is only minimal dilation of the internal ring, the hernia repair can be completed by placing a few interrupted sutures at the medial border of the internal ring and completing a Bassini repair.
6. If the hernia is moderately sized, use a modification of the Bassini repair. Place a series of interrupted sutures in the transversalis fascia beginning medially at the level of the pubic tubercle, and carry the sutures laterally as far as the medial border of the internal ring. Incorporate the posterior fibers of the conjoint tendon, or use the posterior fascia of the transversalis abdominous muscle with some of the muscular fibers of the internal oblique muscle.
7. Begin suturing medially at Cooper's ligament, and transition at the femoral sheath to Poupart's ligament. Place the sutures 1 cm apart, and use nonabsorbable monofilament suture of 00 or heavier. Tie the sutures to reinforce the floor of the inguinal canal.
8. The reconstructed deep inguinal ring should be tight enough to prohibit protrusion of a new hernial sac without obstructing the blood supply of the cord or venous return of the testicle.
9. Replace the ilioinguinal nerve and the spermatic cord in the inguinal canal, and reapproximate the external oblique aponeurosis over the cord with interrupted, nonabsorbable, 000 sutures, taking care not to damage the underlying nerve or cord.
10. Close the subcutaneous tissues with interrupted, fine, absorbable sutures in Scarpa's fascia, and close the skin with staples or subcuticular sutures. Apply a sterile dressing.

II. Surgical Repair of Direct Inguinal Hernias

1. The skin incision is the same as for repair of the indirect inguinal hernia. The direct inguinal hernia appears as a diffuse bulge in the area of Hesselbach's triangle, appreciated by palpating with a fingertip. Reduce the direct inguinal hernia bulging through Hesselbach's triangle with a series of interrupted, inverting, 00, nonabsorbable sutures placed in the redundant properitoneal fat. It is not necessary to provide high ligation and amputation of the hernial sac unless the sac is narrow-necked or diverticular, in which case the sac may be dealt with as described for the repair of the indirect hernia.
2. Repair the fascial defect in Hesselbach's triangle with a Cooper's ligament or modified McVay-type repair. Use sharp and blunt dissection of the floor of the inferior portion of the inguinal canal to

expose the lacunar ligament and Cooper's ligament. Dissect laterally along Cooper's ligament as far as the medial aspect of the femoral vein. Make a relaxing incision in the deep portion of the anterior rectus sheath, then grasp the medial and superior edge of the defect in Hesselbach's triangle with Allis clamps.

3. Place a series of interrupted sutures, beginning medially at the pubic tubercle, and carry laterally as far as the femoral vein. Place these sutures first in the periosteum of the pubic tubercle, and then more laterally in Cooper's ligament itself. With the superior bite of the suture, catch the superior edge of the hernia defect. Place 4 to 8 sutures. Shift the suture line anteriorly, continuing the lateral portion of the repair by suturing the remaining portion of the superior edge of the defect in Hesselbach's triangle to the inguinal ligament ("transition" stitch).
4. Check the appropriate snugness of the deep inguinal ring. When all sutures are placed, tie the sutures medial to lateral. Check the hernia repair for sturdiness and for excessive tension on the sutures. Replace the cord and the ilioinguinal nerve in the bed of the inguinal canal, and reapproximate the external oblique aponeurosis over these structures. Close the subcutaneous tissue and skin, and apply a sterile dressing. Close the skin with microporous adhesive tape or a subcuticular suture.

Hematemesis and Upper Gastrointestinal Bleeding

I. Clinical Evaluation

- A. Determine the duration of hematemesis (vomiting bright red blood or coffee ground material), volume of blood, recent hematocrit. Determine whether the bleeding occurred after forceful vomiting (Mallory-Weiss Syndrome).
- B. Abdominal pain, melena, hematochezia (bright red blood per rectum); history of peptic ulcer, esophagitis, prior bleeding episodes may be present.
- C. **Precipitating Factors:** Use alcohol, aspirin, nonsteroidal anti-inflammatory agents, steroids, anticoagulants should be sought.
- D. **Past Testing or Treatment:** X-ray studies, endoscopy.
- E. The patient may have a past history of gastrointestinal bleeding; prior operations, cardiovascular disease, bleeding disorders.

II. Physical Exam

- A. **General:** Pallor, shallow rapid respirations; tachycardia indicates a 10% blood volume loss; postural hypotension with an increase pulse of 20 and a decrease in systolic of 20 indicates a 20-30% loss.
- B. **Skin:** Delayed capillary refill, pallor. Stigmata of liver disease: Jaundice, spider angiomas, parotid gland hypertrophy.
- C. **Chest:** Gynecomastia (cirrhosis).
- D. **Abdomen:** Scars, tenderness, masses, hepatomegaly; dilated abdominal veins. Stool gross or occult blood.

E. Depressed mental status.

III. Laboratory Evaluation

- A. CBC, SMA 12, liver function tests, amylase, PT/PTT, type and cross PRBC, FFP. CBC q6h.
- B. EKG, UA. CXR, upright abdomen (evaluate for free air under the diaphragm).

IV. **Differential Diagnosis of Upper Bleeding:** Peptic ulcer, gastritis, esophageal varices, Mallory Weiss tear (gastroesophageal junction tear due to vomiting or retching), esophagitis, swallowed blood from epistaxis, malignancy (esophageal, gastric), angiodysplasias, aorto-enteric fistula, hematemesis.

V. Diagnostic and Therapeutic Approach to Upper Gastrointestinal Bleeding

- A. If the bleeding appears to have stopped or has significantly slowed, medical therapy with H₂ blockers, and saline lavage is usually all that is required. The diagnostic and therapeutic approach is based on an estimate of the volume and duration of blood loss, and on the patient's clinical history.
- B. Place a minimum of two 14-16 gauge IV lines. Administer 1-2 liters of lactated Ringer's solution until blood is ready, then transfuse PRBC's as fast as possible; estimate blood transfusion requirement based on blood loss rate, vital signs (typically 2-6 units PRBC's are needed). Administer type O negative blood in emergent situations. If hypotensive consider endotracheal intubation.
- C. Rapidly assess the patient's need for surgical intervention. Central venous monitoring or pulmonary artery catheter monitoring is necessary in the presence of hypotension or hypoxemia.
- D. Place a large bore nasogastric tube, then lavage with 2 L of room temperature tap water, then connect to low intermittent suction, repeat lavage q1h.
- E. Administer oxygen by nasal cannula, guided by pulse oximetry. Foley to closed drainage. Keep patient NPO.
- F. Record volume and character of lavage. The NG tube may be removed when bleeding is no longer active.
- G. If bright red blood is present in nasogastric tube aspirate, lavage with room temperature saline to remove blood clots.
- H. Check serial hematocrits, and maintain greater than 30 vol%. Monitor for coagulopathy and correct if necessary with fresh frozen plasma. Consider pulmonary artery catheterization (Swan-Ganz) to assess effectiveness of resuscitation.
- I. Definitive diagnosis requires upper endoscopy at which time electrocoagulation of bleeding sites may be completed.

VI. Mallory-Weiss Syndrome

- A. This disorder is characterized by a mucosa tear at the gastroesophageal junction. It frequently follows violent retching and vomiting.
- B. Treatment is supportive, and the majority of patients stop bleeding spontaneously. Endoscopic coagulation or operative suturing may rarely be necessary.

VII. Medical Treatment of Peptic Ulcers or Gastritis

- Ranitidine (Zantac) 50 mg IV bolus, then continuous infusion at 6.25-12.5 mg/h [150-300 mg in 500 mL D5W over 24h (21 cc/h)], or 50 mg IV q6-8h **OR**
- Cimetidine (Tagamet) 300 mg IV bolus, then continuous infusion at 37.5-50 mg/h (900 mg in 500 cc D5W over 24h), or 300 mg IV q6-8h **OR**
- Famotidine (Pepcid) 20 mg IV q12h.

Variceal Bleeding

Hemorrhage from esophageal and gastric varices is a severe complication of chronic liver disease.

I. Clinical Evaluation

- A.** Variceal bleeding should be considered in any patient who presents with significant upper gastrointestinal bleeding, because some patients with liver disease do not exhibit the classic signs (eg, spider angiomas, palmar erythema, leukonychia, clubbing, parotid enlargement, Dupuytren's contracture).
- B.** When patients present with jaundice, lower extremity edema and ascites, the diagnosis of decompensated liver disease is obvious.
- C.** The severity of the bleeding episode can be assessed on the basis of the presence of orthostatic changes (eg, resting tachycardia, postural hypotension), which indicates that about one third or more of the patient's blood volume has been lost.
- D.** If the patient's sensorium is altered due to hepatic encephalopathy, the high risk of aspiration mandates endotracheal intubation and protection of the airway.
- E.** Placement of a large-caliber nasogastric tube (22 F or 24 F or, rarely, an Ewald tube for massive bleeding) permits lavage for removal of luminal blood and clots in preparation for endoscopy.
- F.** Lavage should be performed with tap water, because the benefit of ice water is dubious and saline may contribute to retention of sodium and water.

II. Resuscitation

- A.** Blood lost through the gastrointestinal tract should be replaced as soon as possible. While blood for transfusion is being made available, intravascular volume should be replenished with intravenous albumin in isotonic saline solution (Albuminar-5) or normal saline solution if the patient does not have ascites or evidence of decompensation.
- B.** Once normovolemia is established, intravenous infusion should be changed to solutions with a lower sodium content (5% dextrose in water or 5% dextrose with 0.45% sodium chloride).
- C.** Fresh frozen plasma is not indicated for volume repletion and it is needed only if massive transfusion is required because of the "washout" phenomenon; in such cases, calcium should also be replaced.
- D.** Blood should be transfused to maintain a hematocrit of at least 25%. Serial hematocrit estimations during continued bleeding are done

primarily to determine whether replacement is adequate. Values may, however, be inaccurate after acute blood loss. Patients must be hemodynamically stable before endoscopy is performed.

III. Treatment of Variceal Hemorrhage

A. Pharmacologic Agents

1. **Octreotide** acetate (Sandostatin) (50 mcg/hr for 48 hours), a somatostatin analog, is beneficial in controlling hemorrhage.
2. **Vasopressin (Pitressin)**, a posterior pituitary hormone, causes splanchnic arteriolar vasoconstriction and reduction in portal pressure.
 - a. Bleeding stops in about half of patients with active upper gastrointestinal bleeding who are given vasopressin. Intravenous dosage is 0.3 U/min; complications increase with dosages of 0.4 to 0.6 U/min.
 - b. **Side effects** include skin pallor, abdominal cramps, decreased intestinal transit, bradycardia, hyponatremia, and antidiuretic hormone effect.
 - c. **Complications:** Angina, myocardial ischemia or infarction, arrhythmia, mesenteric ischemia or infarction, ischemia of the extremities, renal insufficiency, cerebrovascular accident, and activation of fibrinolysis.
 - d. Concomitant use of transdermal nitroglycerin, 0.4 mg/hr for 12 hours may reduce portal pressure by about 10%. This agent mitigates the vasoconstrictor effects of vasopressin on the myocardial and splanchnic circulations. Intravenous nitroglycerin should be avoided because of hypotension.

- B. Additional treatments should be considered if bleeding continues, as confirmed by fresh blood aspirate from the nasogastric tube and the need for continued blood transfusion (2 to 3 U of packed cells every 8 hours after start of infusion).

C. Tamponade Devices

1. Bleeding from varices can be reduced with the use of tamponade balloon tubes. However, the benefit is temporary, and prolonged tamponade causes severe esophageal ulceration, limiting the option of subsequent endoscopic therapy.
2. The Linton-Nachlas tube is recommended; it has a gastric balloon and several ports in the esophageal component that allow use of suction. The tube is kept in place for 6 to 12 hours while preparations for endoscopic or radiologic treatment are being made.

D. Endoscopic Management of Bleeding Varices

1. Endoscopic sclerotherapy involves injection of a sclerosant solution into varices during endoscopy. The success of the treatment is enhanced by repeating treatments at weekly intervals or less often.
2. **Sclerotherapy** has been shown to control actively bleeding varices in more than 90% of cases. Because portal pressure is not significantly affected by endoscopic sclerotherapy, varices do reform; thus, yearly follow-up is needed.
3. **Endoscopic variceal ligation** involves placement of tiny rubber bands on varices during endoscopy.
 - a. There are fewer complications with ligation than with sclerotherapy, and bleeding and mortality rates are lower.

- b. Ligation is the preferred treatment when varices are large and there is no active bleeding.

E. Surgery

1. Portal-systemic shunt surgery is the last resort and the most definitive therapy for bleeding varices. The placement of a shunt creates an anastomosis between portal and systemic veins, allowing decompression of the hypertensive portal venous system and almost complete elimination of rebleeding. However, the procedure has a 30-40% rate of hepatic encephalopathy, and there is no difference in survival rates between shunt surgery and medical or nonendoscopic treatments.
2. Only patients with well-compensated liver disease are considered surgical candidates.
3. The distal splenorenal shunt is preferred. This shunt is placed between the distal splenic vein and the left renal vein.
4. Portacaval shunt surgery is easier to perform and may be preferable in an emergency or with severe decompensation.

F. Radiologic Procedures

1. **Transjugular intrahepatic portacaval shunt (TIPS):** Under fluoroscopy, a needle is advanced into the liver through the internal jugular and hepatic veins and inserted into a large branch of the portal vein. A balloon is then used to enlarge the track to permit the placement of a stent. Good blood flow through the stent results in satisfactory decompression of collaterals and cessation of bleeding.
2. Because TIPS is a shunt procedure, encephalopathy occurs in about 35% of patients and occlusion of the stent may cause recurrent variceal bleeding. TIPS is useful for patients who are awaiting liver transplantation after unsuccessful endoscopic therapy.

IV. Approach to Treatment of Variceal Hemorrhage

- A. Patients initially should be given vasopressin infusion and transdermal nitroglycerin while awaiting endoscopic treatment.
- B. If bleeding is not brisk and varices are large, endoscopic ligation is preferred; for active bleeding from a spurting varix, sclerotherapy is best.
- C. Subsequent ligation or sclerotherapy should be performed weekly until varices are obliterated. Endoscopy is repeated at monthly, 3- to 6-month, and yearly intervals for as long as the patient is alive.
- D. Treatment failure warrants the use of TIPS or portal-systemic shunt.
- E. Liver transplantation should be considered in potential candidates when therapy for variceal bleeding fails.

Peptic Ulcer Disease

I. Clinical Evaluation

- A. Of the three well-identified causes of peptic ulcer disease--*Helicobacter pylori*, nonsteroidal anti-inflammatory drugs (NSAIDs), and pathologically high acid-secreting states (Zollinger-Ellison syndrome)--*H pylori* is the most frequent cause.
- B. H₂ blockers, sucralfate (Carafate), omeprazole (Prilosec) only temporarily heal ulcers. However, eradication of *H pylori* permanently

- cures peptic ulcer disease, eliminating the need for long-term therapy.
- C.** H pylori is found in almost all cases of chronic duodenal ulcers, and in the majority of cases of gastric ulcers that are not caused by use of an NSAID.
- D. Nonendoscopic Tests for Helicobacter pylori:**
1. If the patient has documented peptic ulcer disease and is having recurrent symptoms, it is reasonable to treat with antibiotics without further study if serologic tests are positive for H pylori. Eradication is usually assessed clinically by alleviation of symptoms. If symptoms recur, a breath test or endoscopic biopsy is necessary to confirm eradication.
 2. A reliable urea breath test is available, in which the patient ingests a capsule containing urea with radiolabeled carbon. The urea is broken down by the H pylori present, and the tagged carbon dioxide is expired via the lungs.
- E. Endoscopic biopsy** and histologic examination are still the primary means of H pylori diagnosis.

II. Treatment of Peptic Ulcer Disease

A. Eradication of H pylori Infection:

1. Treat duodenal and gastric ulcers, new ulcers, recurrent ulcers, and maintained ulcers with antimicrobials.
 2. **Triple Drug Therapy:**
 - Bismuth Subsalicylate (Pepto-Bismol) 2 tabs or 30 mLs PO qid
 - AND**
 - Metronidazole (Flagyl) 250 mg PO qid x 14 days **AND**
 - Tetracycline 500 mg qid x 14 days.
 3. **Omeprazole and Clarithromycin Therapy:**
 - Clarithromycin (Biaxin) 500 mg PO tid x 14 days **AND**
 - Omeprazole (Prilosec) 40 mg PO qd x 14 days, then 20 mg PO qd x 14 days.
 4. **Omeprazole and Antibiotic Therapy:**
 - Omeprazole (Prilosec), 40 mg qd **AND** tetracycline 500 mg qid or amoxicillin, 500 mg PO qid, for 14 days.
- B. H2 Blockers:** H2 blockers may be used in either twice-daily or single night-time doses.
1. **Dosages for Acute Therapy:**
 - Cimetidine (Tagamet), 400 mg bid or 800 mg hs
 - Ranitidine (Zantac), 150 mg bid or 300 mg hs
 - Famotidine (Pepcid), 20 mg bid or 40 mg hs
 - Nizatidine (Axid Pulvules), 150 mg bid or 300 mg hs
 2. Side effects are uncommon; occasionally drug interactions, neurologic symptoms.
- C. Proton Pump Inhibitors:**
1. **Omeprazole (Prilosec):** Reserved for ulcers refractory to H2 blockers and sucralfate. 20 mg qd. Side effects are rare.
 2. **Lansoprazole (Prevacid):** 15 mg before meals [15, 30 mg]
- D. Mucosal Protective Agents:**
1. **Sucralfate (Carafate):**
 - a. Aluminum-containing, mucosal, protective agent; 1 gm before meals and hs.
 - b. Constipation is a common. Can bind with other drugs.

2. Misoprostol (Cytotec):

- a. 200 mcg PO qid; prostaglandin E1 analogue; mucosal protective.
- b. Not a first-line agent.
- c. Effective in healing peptic ulcer disease; side effects are significant; 25% experience diarrhea that resolves by reducing the dosage to 200 mcg bid. Can cause spontaneous abortion.

III. Maintenance Therapy for Peptic Ulcer Disease

- A. If *H. pylori* therapy has been completed, most ulcers will not recur, and maintenance therapy will not be required.
- B. **Maintenance dose is half of the standard therapeutic dose:**
 - Cimetidine (Tagamet), 400 mg hs
 - Ranitidine (Zantac), 150 mg hs
 - Famotidine (Pepcid), 20 mg hs
 - Nizatidine (Axid Pulvules), 150 mg hs
 - Sucralfate (Carafate), 1 g bid

IV. Surgical Treatment of Peptic Ulcer Disease

- A. **Indications for Surgery:** Exsanguinating hemorrhage, >5 units transfusion in 24-hours, rebleeding during same hospitalization. Intractability, perforation, gastric outlet obstruction, endoscopic signs predictive of rebleeding (visible vessel).
- B. **Emergency Surgery for Peptic Ulcer Disease**
 - 1. **Unstable Patients** should receive a truncal vagotomy, oversewing of bleeding ulcer bed, and a pyloroplasty.
 - 2. **Stable Patients** should be managed with oversewing of the ulcer bed with either a vagotomy and antrectomy or a proximal gastric vagotomy.
- C. **Surgical Therapy of Duodenal Ulcer Disease**
 - 1. **Operative Therapy:** If the ulcer is still active after 12 weeks of medical therapy, surgery should be considered.
 - 2. **Vagotomy Procedures**
 - a. Truncal vagotomy with drainage procedure (pyloroplasty or gastrojejunostomy), or
 - b. Selective vagotomy with drainage procedure, or
 - c. Proximal gastric (parietal cell, highly selective) vagotomy (usually without drainage procedure).
 - 3. **Vagotomy and Antrectomy** with gastroduodenostomy (Billroth I) or gastrojejunostomy (Billroth II). Antrectomy consists of removal of distal one third of stomach.
 - 4. **Vagotomy Procedures**
 - a. **Selective Vagotomy:** Division of vagal branches, denervating stomach but preserving innervation of liver and biliary tree and celiac ganglia. Drainage procedure is necessary to allow passage of food through the denervated pylorus.
 - b. **Proximal Gastric Vagotomy:** Division of vagal branches to the parietal region of the stomach with preservation of vagal branches to the antrum and pylorus; a drainage procedure is not necessary because innervation of pylorus is left intact.
- D. **Gastric Ulcer Treatment**
 - 1. Six-week trial of H₂ blockers if biopsy negative.
 - 2. *Helicobacter pylori* treatment (see above).

- 3. Indications for Surgery:** Documented, incomplete healing following medical therapy for 6-12 weeks; acute hemorrhage, obstruction, perforation, gastric cancer.
- 4. Surgical Treatment:** Gastric resection to include ulcer. Vagotomy should be added in patients with increased acid secretion.
- 5. Adjunctive Measures:** Discontinue foods or medications which worsen symptoms, such as alcohol, smoking, aspirin, caffeine, glucocorticoids. Eliminate Precipitating Factors such as smoking, alcohol, drugs.

Gastric Cancer

I. James Sarfeh, M.D.

I. Clinical Evaluation

- A. History:** Early gastric carcinoma is usually asymptomatic. May present with pain, indigestion (especially if >40 years old), anorexia, weight loss, nausea, vomiting, abdominal mass, dysphagia.
- B.** Occult blood in stool and hematemesis may sometimes occur, fatigue, weight loss, anemia, ascites.
- C. Risk Factors:** Diet consisting of smoked, salted, pickled foods, nitrosamines; smoking, alcohol. Genetic factors, previous gastric surgery, pernicious anemia, polyps, atrophic gastritis, hypertrophic gastropathy (Menetrier's disease).
- D. Laboratory Evaluation:** Endoscopy with multiple biopsies and brushing, lavage cytology. CT scan of abdomen for staging.

II. Staging of Gastric Cancer

TNM Classification:

Primary Tumor (T)

- | | |
|----|---|
| T1 | Tumor limited to mucosa and submucosa |
| T2 | Tumor involves mucosa, submucosa and extends to or into serosa, but not penetrating serosa. |
| T3 | Penetration through serosa, but no invasion of contiguous structures. |
| T4 | Penetration through mucosa and invasion of contiguous structures. |

Nodal Involvement (N)

- | | |
|----|---|
| N0 | No metastases to regional lymph nodes. |
| N1 | Perigastric lymph node involvement within 3 cm of primary tumor along the lesser or greater curvature. |
| N2 | Regional lymph node involvement, more than 3 cm from primary tumor, removable at operation; including nodes located along the left gastric, splenic, celiac, and common hepatic arteries. |
| N3 | Intra-abdominal lymph node involvement that is not removable at operation. |

Distance Metastasis (M)

- | | |
|----|------------------------------|
| M0 | No distant metastases known. |
| M1 | Distant metastases present. |

Surgical result (R)

- R0 - No residual tumor.
- R1 - Microscopic residual tumor.
- R2 - Macroscopic residual tumor.

III. Treatment of Gastric Cancer

- A. Subtotal gastrectomy is used for palliation of advanced cancer.
- B. Extended subtotal or complete gastrectomy with removal of regional nodes may be curative for localized cancers.
- C. Esophagogastrectomy is indicated for adenocarcinoma of the cardia and esophageal junction.
- D. Chemotherapy and radiation therapy are given for recurrence.

Gastroesophageal Reflux Disease

I. Clinical Evaluation of Gastroesophageal Reflux Disease (GERD)

- A. The most common symptom is heartburn, a burning sensation in the epigastric or retrosternal area, rising toward the throat, often occurring postprandially.
- B. Regurgitation, dysphagia, and belching may occur.
- C. Hoarseness, nocturnal cough and wheezing may be caused by chronic reflux. Asthma may be exacerbated by gastroesophageal reflux.
- D. Chronic reflux may cause Barrett's esophagus (columnar metaplasia of esophageal mucosa), and may predispose to esophageal adenocarcinoma.
- E. GERD is associated with decreased lower esophageal sphincter pressure caused by inappropriate relaxation of the lower esophageal sphincter, low basal sphincter pressure, or increases in intra-abdominal pressure. Sphincter tone can be impaired by consumption of fatty foods and anticholinergic medications.

II. Antireflux Therapy

- A. **Empiric Therapy:** With classic symptoms (heartburn, regurgitation), and no evidence of complications or systemic illness, a presumptive diagnosis of reflux disease may be made. An H₂ receptor blocker and prokinetic drug may be prescribed.
- B. If reflux symptoms persist after 8-12 weeks of treatment, endoscopy is indicated.
- C. Severe or atypical symptoms occurring many times a week warrant early diagnostic evaluation with esophagoscopy. 24-hour ambulatory esophageal pH monitoring is the "gold standard" of testing.

III. Non-Pharmacologic Therapy for Gastroesophageal Reflux Disease

- A. **Inclined Bed:** Place blocks (6 inches high) under head of bed or support patient's upper body with a foam wedge.
- B. **Dietary Modifications:**
 - 1. Avoid foods that reduce lower esophageal sphincter pressure, impair gastric emptying, or that irritate mucosal lining. Chocolate and carminatives (spearmint, peppermint), fatty foods, onions may exacerbate reflux. Coffee and tomato-based products irritate the esophageal mucosa.

2. Refrain from overeating, and avoid eating before bedtime.
3. **Avoid causative medications** such as anticholinergic drugs, potassium chloride, nonsteroidal anti-inflammatory drugs, theophylline, calcium channel blockers, and progesterone. Prolonged contact of the esophagus with corrosive drug tablets or capsules may irritate mucosa. When taking medications, patients should consume ample liquid.

C. Pharmacologic Treatment of Gastroesophageal Reflux Disease

Agent:	Dosage:	Mechanism of action:
Histamine-2 blockers Cimetidine (Tagamet) Ranitidine (Zantac) Famotidine (Pepcid) Nizatidine (Axid Pulvules)	400-800 mg bid 150 mg bid 20-40 mg bid 150 mg bid	Inhibit gastric acid secretion
Prokinetic drugs Bethanechol chloride Metoclopramide (Reglan) Cisapride (Propulsid)	Up to 25 mg qid 10 to 15 mg up to four times daily Up to 20 mg qid [10 mg]	Increase lower esophageal sphincter pressure, increase peristalsis Neurologic and psychotropic side effects. Does not have neurologic or psychotropic side effects
H⁺,K⁺-ATPase (proton-pump) inhibitors Omeprazole (Prilosec) Lansoprazole (Prevacid)	20 mg qd or bid 30 mg qd	Inhibit gastric acid secretion

IV. Refractory Cases

- A. Doubling the standard dose or the dose frequency of medications may provide greater symptomatic relief and a higher rate of healing.
- B. Consider substituting omeprazole (Prilosec), which profoundly inhibits gastric acid production.
- C. **Anti-Reflux Surgery:** A small minority of patients with truly intractable disease should be considered for antireflux surgery to reestablish a competent lower esophageal sphincter. Antireflux surgery has shown disappointing long-term results, and is indicated only on rare occasions.
- D. **Surgical Therapy**
 1. Indicated if patient fails medical therapy or if severe esophagitis or complications.
 2. Surgery is indicated for Type II, III, IV hernias.
 3. **Nissen Fundoplication:** Fundus is completely wrapped around esophagus; hiatus closed; may be done laparoscopically. Most commonly used operation.
 4. **Hill Procedure:** Fundic wrap and anchoring to pre-aortic fascia; hiatus closed.
 5. **Belsey Procedure:** Exaggerated gastroesophageal junction angle in the stomach is anchored below diaphragm. Usually done via a transthoracic approach.
 6. **Collis Gastroplasty:** A "tube" of gastric mucosa is created. Useful for severe strictures.

7. **Laparoscopic Repair:** A simplified Nissen fundoplication approach.

Lower Gastrointestinal Bleeding

J. Craig Collins, M.D.

The spontaneous remission rate for lower gastrointestinal bleeding, even with massive bleeding, is 80% (the same as for upper gastrointestinal bleeding). No source of bleeding can be identified in 12%, and bleeding is recurrent in 25%.

Lower GI bleeding is usually intermittent, regardless of cause. Bleeding has usually ceased by the time the patient presents to the emergency room, although copious amounts of blood and clots may continue to be passed from the rectum.

I. Initial Clinical Evaluation

- A. The severity of blood loss and hemodynamic status should be assessed immediately. Initial management consists of resuscitation with colloidal solutions (hetastarch [Hespan]) or crystalloid solutions (sodium chloride) and with blood products if necessary.
- B. An initial diagnostic evaluation, to determine the source of bleeding, is performed while the patient is being resuscitated.
- C. The duration and quantity of bleeding are assessed; however, the duration of bleeding is often underestimated and the quantity is often overestimated.
- D. Risk factors that may have contributed to the bleeding should be assessed, such as the use of nonsteroidal anti-inflammatory drugs, anticoagulants, history of colonic diverticulosis, renal failure, coagulopathy, colonic polyps, hemorrhoids, chemotherapy or radiotherapy.
- E. **Hematochezia:** Bright red or maroon blood per rectum suggests a lower GI source; however, 11-20% of patients with an upper GI bleed will have hematochezia as a result of rapid blood loss (these patients are usually in shock).
- F. **Melena:** Sticky, black, foul-smelling stools suggest a source proximal to the ligament of Treitz, but can result from bleeding in the small intestine or proximal colon. Iron and bismuth can turn stools black but not melanotic (shiny and tarry).
- G. **Malignancy** may be indicated by a change in stool caliber, anorexia, weight loss and malaise.
- H. Patients may have a history of hemorrhoids, diverticulosis, inflammatory bowel disease, peptic ulcer, gastritis, cirrhosis or esophageal varices.

I. Associated Findings

- 1. **Abdominal pain** may result from ischemic bowel, inflammatory bowel disease, or a ruptured aortic aneurysm.
- 2. **Painless, massive bleeding** often indicates vascular bleeding from diverticuli, angiodysplasia or hemorrhoids.
- 3. **Bloody diarrhea** suggests inflammatory bowel disease or an infectious origin.
- 4. **Bleeding with rectal pain** is seen with anal fissures, hemorrhoids, and rectal ulcers.

5. **Chronic constipation** suggests hemorrhoidal bleeding; new onset constipation or thin stools suggests a left-sided colonic malignancy.
6. **Blood on the toilet paper or dripping** into the toilet water after a bowel movement indicates a perianal source.
7. **Blood coating the outside of stool** suggests a lesion in the anal canal.
8. **Blood streaking or mixed in with the stool** may result from a polyp or malignancy in the descending colon.
9. **Maroon colored stools** often indicate small bowel and proximal colon bleeding.

II. Physical Examination

- A. **Postural Hypotension** suggests a 20% blood volume loss; whereas, signs of shock, pallor, hypotension, and tachycardia indicate a 30-40% blood loss.
- B. The skin may be cool and pale with delayed capillary refill if bleeding has been significant.
- C. Stigmata of liver disease including jaundice, caput medusae, gynecomastia, and palmar erythema should be sought since these patients frequently have GI bleeding.
- D. Cutaneous manifestations of the Rendu-Osler-Weber syndrome (telangiectasias) or blue rubber bleb nevus syndrome (blue nevi) should be sought.

III. Approach to Diagnosis of Lower Gastrointestinal Bleeding

- A. Rapid clinical evaluation and resuscitation should precede diagnostic or therapeutic studies. Intravenous fluids (1-2 liters) should be infused over 10-20 minutes to restore intravascular volume, and blood is transfused if there is rapid ongoing blood loss or if hypotension or tachycardia is present. Coagulopathy is corrected with fresh frozen plasma or platelets.
- B. When small amounts of bright red blood are passed per rectum, the lower gastrointestinal tract can be assumed to be the source.
- C. In patients with large-volume maroon stools, nasogastric tube aspiration should be performed to exclude massive upper gastrointestinal hemorrhage. Occult blood testing of lavage fluid is useless because mild trauma from tube placement may cause a positive result.
- D. If the nasogastric aspirate contains no blood, then anoscopy and sigmoidoscopy should be performed to determine whether a colonic mucosal abnormality (ischemic or infectious colitis) or hemorrhoids might be the cause of bleeding.
- E. If these measures fail to yield a diagnosis, rapid administration of polyethylene glycol-electrolyte solution (CoLyte) should be initiated orally or by means of a nasogastric tube; 4 L of the lavage solution is infused over a 2- to 3-hour period. This allows diagnostic and therapeutic colonoscopy and adequately prepares the bowel should emergency surgery become necessary.

IV. Causes of Lower Gastrointestinal Bleeding

- A. Angiodysplasia and diverticular disease of the right colon account for the vast majority of episodes of acute lower gastrointestinal bleeding.
- B. Most acute LGI bleeding originates from the colon; however, 15-20% of

episodes arise from the small intestine and the upper gastrointestinal tract.

Differential Diagnosis of Lower GI Bleeding

Diverticulosis	presence of an underlying structural lesion
Angiodysplasia (vascular ectasia)	
Colonic polyp	Neutropenic typhlitis
Carcinoma of the colon	Tuberculosis
Radiation colitis	Crohn's disease
Hemorrhoids	
Ischemic colitis	
Stercoral ulcer	
Ulcerative colitis	
Coagulopathy (or anticoagulation) in the	

C. Elderly Patients: Diverticulosis and angiodysplasia are the most common causes of lower GI bleeding.

D. Younger Patients: Hemorrhoids, anal fissures, and inflammatory bowel disease (IBD) are more common causes.

E. Angiodysplasia

1. Angiodysplastic lesions are small vascular tufts that are formed by capillaries, veins, and venules.
2. Angiodysplastic lesions are commonly noted during colonoscopy, appearing as red dots or spider-like lesions 2 to 10 mm in diameter.
3. Angiodysplastic lesions develop secondary, to chronic colonic distention, which leads to obstruction of venules.
4. Angiodysplastic lesions are associated with advanced age, and they have a prevalence rate of 25% in elderly patients. These lesions have also been associated with chronic renal failure, CREST syndrome, Rendu-Osler-Weber syndrome, and cirrhosis. Their association with aortic stenosis remains to be established.
5. Even though angiodysplasia may be present throughout the entire colon, the most common site of bleeding remains the right colon. Most patients with angiodysplasia have recurrent minor bleeding; however, massive bleeding is not uncommon.

F. Diverticular Disease

1. Diverticular disease is the most common cause of acute lower gastrointestinal bleeding.
2. 60% to 80% of bleeding diverticula are located in the right colon.
3. 90% of all diverticula are found in the left colon.
4. Diverticular bleeding tends to be massive, but it stops spontaneously in 80% of patients with only supportive care, and the rate of rebleeding is only 25%.

V. Definitive Management of Lower GI Bleeding

A. Colonoscopy

1. Colonoscopy is the procedure of choice for diagnosing colonic causes of gastrointestinal bleeding. It should be performed after adequate preparation of the bowel, which permits identification of 80% of all causative colonic lesions. If the bowel cannot be adequately prepared because of persistent, acute bleeding, a bleeding scan or angiography is preferable.
2. Endoscopy may be therapeutic for angiodysplastic lesions, polyps, and tumors, which can be effectively coagulated.
3. If colonoscopy fails to reveal a source for the bleeding, the patient should be observed, since in about 80% of cases, bleeding ceases spontaneously.

B. Bleeding Scan

1. The technetium-labeled ("tagged") red blood cell bleeding scan can detect bleeding sites when bleeding is intermittent.
2. If the result is positive, the next step is colonoscopy or angiography.

C. Angiography

1. Selective mesenteric angiography detects arterial bleeding that occurs at a rate of 0.5 mL/min or faster.
2. Diverticular bleeding classically causes pooling of contrast medium within a specific diverticulum (extravasation).
3. Bleeding angiodysplastic lesions demonstrate abnormal vasculature.
4. Bleeding from angiodysplastic lesions usually is slow and rarely necessitates therapeutic intervention. However, when active bleeding is seen with diverticular disease or angiodysplasia, selective arterial infusion of vasopressin is effective in arresting hemorrhage in 90%.

D. Evaluation of the Small Bowel

1. If bleeding persists but no source is noted, the small bowel must be considered as a possible source.
2. The preferred technique for evaluating the small bowel is push enteroscopy (with an overtube) in combination with enteroclysis barium study.
3. Meckel's diverticulum, which usually presents in younger patients, is a common site of bleeding in the small intestine. The diagnosis can be confirmed by radionuclide Meckel's scanning, which identifies the ectopic gastric mucosa.

E. Surgery

1. If bleeding continues and no source has been found, surgical intervention is warranted.
2. Surgical resection may be indicated for patients with recurrent diverticular bleeding, or for patients who have had persistent bleeding from colonic angiodysplasia and have required blood transfusions.
3. Treatment of lower gastrointestinal bleeding involves resection of the involved segment--for example, a right hemicolectomy or sigmoid resection. Patients with a diffuse process, such as ulcerative colitis, require a total proctocolectomy with ileostomy.
4. When laparotomy fails to identify a definitive source of bleeding, intraoperative endoscopy may be a useful adjunct.

VI. Colon Polyps and Colon Cancers

- A. These rarely cause significant acute LGI hemorrhage.
- B. Left-sided and rectal neoplasms are more likely to cause gross bleeding than right sided lesions. Right sided lesions are more likely to cause anemia and occult bleeding.
- C. **Diagnosis:** Colonoscopy.
- D. **Treatment:** Colonoscopic excision or surgery.

VII. Inflammatory Bowel Disease

- A.** Ulcerative colitis can occasionally cause severe GI bleeding associated with abdominal pain and diarrhea.
- B. Diagnosis:** Colonoscopy and biopsy.
- C. Treatment:** Medical treatment of the underlying disease; surgery is required on rare occasions.

VIII. Ischemic Colitis

- A.** This disorder is seen in elderly patients with known vascular disease; abdominal pain may be postprandial, and is associated with bloody diarrhea or rectal bleeding. Severe blood loss is unusual but can occur.
- B. Diagnosis:** Abdominal films may reveal "thumbprinting", caused by submucosal edema. Colonoscopy reveals a well-demarcated area of hyperemia, edema, and mucosal ulcerations. The splenic flexure and descending colon are the most common sites.
- C. Treatment:** Most episodes resolve spontaneously; however, vascular bypass or resection may be required.

IX. Hemorrhoids

- A.** Hemorrhoids rarely cause massive acute blood loss. In patients with portal hypertension, rectal varices must be sought.
- B. Diagnosis:** Anoscopy and sigmoidoscopy.
- C. Treatment:** High fiber diet, stool softeners, or hemorrhoidectomy.

X. Small-Bowel Sources of Bleeding

- A.** The small bowel accounts for only 5% of LGI bleeding, and causes include malignancy, vascular lesions, Crohn's disease, and aortoenteric fistula. Meckel's diverticulum bleeding usually occurs in children but has been reported in adults.
- B. Diagnosis:** Endoscopy, enteroclysis, angiography, and intraoperative enteroscopy.

Anorectal Disorders

I. Hemorrhoids

- A.** Hemorrhoids are dilated veins located beneath the lining of the anal canal.
- B.** Internal hemorrhoids are located in the upper anal canal. External hemorrhoids are located in the lower anal canal.

II. Internal Hemorrhoids

- A.** Internal hemorrhoids become symptomatic when disruption of the supporting tissues allows prolapse and excessive engorgement of the vascular anal cushions. Disruption results from mechanical shearing forces of defecation.
- B.** The most common symptom of internal hemorrhoids is painless rectal bleeding, which is usually bright red and varies from a few drops to a spattering stream at the end of defecation.
- C.** If internal hemorrhoids remain prolapsed outside the anal canal after defecation, dull aching may occur.
- D.** Blood and mucus stains may appear on underwear. Itching in the perianal region is common.

Classification of Internal Hemorrhoids

Grade	Description	Symptoms
1	Nonprolapsing	Minimal bleeding
2	Prolapse with straining, reduce when spontaneously prolapsed	Bleeding, discomfort, pruritus
3	Prolapse with straining, manual reduction require when prolapsed	Bleeding, discomfort, pruritus
4	Cannot be reduced when prolapsed,	Bleeding, discomfort, pruritus

E. Management of Internal Hemorrhoids

- Grade 1 and uncomplicated grade 2 hemorrhoids** may be treated with avoidance of nonsteroidal anti-inflammatory drugs and dietary modification (increased fiber and fluids and avoidance of binding, spicy, and fatty foods).
- Symptomatic grade 2 and grade 3 hemorrhoids**
 - These hemorrhoids are treated with hemorrhoid banding. Through an anoscope, a rubber band ligature is placed above and around the hemorrhoid. The tissue encompassed by the band sloughs in about a week, leaving an ulcer at the site of the banding. Two or three bandings are often required, which are usually performed in separate sessions.
 - Major complications of this procedure are rare and consist of excessive pain, bleeding during sloughing of the band, and infection. Discomfort after banding is usually relieved by warm baths and benzodiazepines if necessary.
- Grade 3 and 4 hemorrhoids** usually require surgical hemorrhoidectomy.

III. External Hemorrhoids

- External hemorrhoids are seen most often in young and middle-aged adults, and they are symptomatic only when they become thrombosed.
 - External hemorrhoids are characterized by rapid onset of constant burning or throbbing pain accompanying a new rectal lump. Bluish skin-covered lumps are easily visible at the anal verge.
 - The pain of an acutely thrombosed hemorrhoid is maximal in the first 48 hours and decreases thereafter to minimal discomfort after the fourth day.
- ### D. Management of External Hemorrhoids
- If patients are seen in the first 48 hours, the entire lesion can be excised easily in an office procedure. Local anesthetic is infiltrated under and around the thrombosed area, and the

thrombus and overlying skin are excised with scissors. The resulting defect heals by secondary intention.

2. If thrombosis occurred more than 48 hours prior, the best approach is to allow spontaneous resolution; symptoms may be relieved by pain medication, sitz baths, and stool softeners. Occasionally, as thrombosed external hemorrhoids resolve, the hematoma may erode through the skin, leading to hematochezia. The problem is self-limiting and requires only hemostasis with direct pressure.

IV. Anal Fissures

- A. Anal fissures are linear ulcers that extend from just below the dentate line to the anal verge. They occur most often in young and middle-aged adults but can affect all ages.
- B. These lesions are most common in the posterior midline but have been reported in the anterior midline in about 10% of patients. Acute anal fissures are cracks in the epithelium, but chronic lesions can cause a skin tag at the outermost edge (the sentinel pile).
- C. Chronic fissures can undergo suppuration and extend into the surrounding tissues, causing a perianal abscess.
- D. Trauma to the anal canal during defecation is the initiating factor for anal fissures.
- E. Anal fissures cause a cutting or tearing anal pain during defecation and of gnawing or throbbing discomfort after defecation. Any associated bleeding is usually slight.
- F. Anal fissure is usually suggested by the history and can be confirmed by inspection of the anus.

G. Treatment of Anal Fissures

1. Medical management:

- a. Bulking fiber supplements together with warm sitz baths and a high-fiber diet are the most helpful treatment methods.
- b. Sitz baths have been shown to decrease resting anal tone, control pain, and promote healing.
- c. Topical medication and suppositories are not effective when used alone but are probably not harmful.

2. Lateral Partial Internal Sphincterotomy

- a. This surgical procedure is indicated for patients in whom medical therapy fails. It consists of surgical division of a portion of the internal sphincter, resulting in a permanent decrease in resting anal tone.
- b. The procedure is highly effective in relieving pain and promoting healing.
- c. **Adverse effects** include incontinence to flatus and seepage.

V. Perianal Abscess

- A. The anal glands, located in the base of the anal crypts at the level of the dentate line, are probably the most common source of perianal infection.
- B. Acute infection presents as an abscess, and chronic infection results in a fistula. Infections start in the intersphincteric plane and can extend upward, downward, and circumferentially around the anus.
- C. The most common symptoms of perianal abscess are swelling and

pain that is throbbing, continuous, and progressive. Fevers and chills may occur. Perianal abscess is common in diabetic and immunosuppressed patients, and there is often a history of chronic constipation or previous abscesses.

D. A tender mass is apparent on rectal exam. Fluctuance or only induration may be apparent.

E. Management of Perianal Abscess

1. A perianal abscess is treated with incision and drainage. Antibiotic therapy alone is not adequate. If the abscess is small, incision and drainage as an office procedure using a local anesthetic is usually possible. If the abscess is large or high in the ischiorectal fossa, regional or general anesthesia may be needed to obtain adequate drainage.

2. A cruciate incision should be made and the corners excised to create an elliptical opening, which promotes drainage and closes more slowly.

3. Antibiotic therapy is required if the patient has systemic signs of infection, is immunocompromised, has a heart murmur, or has a prosthetic heart valve or other intravascular or joint prosthesis. An agent effective against bowel flora, such as Zosyn, Timentin, Cefotetan, is used.

4. Inadequate drainage can lead to necrotizing fasciitis of the perineum, especially in diabetic or immunocompromised patients.

F. An anorectal abscess can progress readily to Fournier's gangrene (necrotizing fasciitis of the perineum and abdominal wall). This life-threatening condition requires immediate, radical debridement.

G. About half of patients with anorectal abscesses will develop a fistula tract between the anal glands and the perianal mucosa, known as a fistula-in-ano, which manifests as either incomplete healing of the drainage site or recurrence. All patients with anorectal abscess should be reexamined 4-6 weeks after treatment of the abscess to determine if a fistula has formed. Permanent healing of an established fistula-in-ano requires a surgical fistulotomy.

VI. Condylomata acuminata

A. Condylomata acuminata, or genital warts, are a common venereal disease caused by the human papillomavirus. They are spread by intimate contact, including autoinoculation, and have an incubation period that ranges from weeks to months.

B. An estimated 1% of sexually active adults have visible lesions. Several subtypes of human papillomaviruses have been associated with development of malignant lesions of the genitalia.

C. Patients with small lesions usually have few symptoms. If lesions become large in size, bleeding, discharge, itching, and pain may occur. Warts are pink or white with a papilliform surface.

D. Treatment of Condylomata

1. Nonsurgical or surgical treatments can be discouraging.

2. Topical caustic agents, such as podophyllum resin or trichloroacetic acid, work best on small lesions when applied repetitively.

3. Lesions that are persistent despite topical treatment, extensive, or that extend into the anal canal are best managed with surgical ablation. Recurrence is common.

Fistula-in-Ano

I. Clinical Evaluation

- A. A fistula-in-ano develops when an anorectal abscess forms a fistula between the anal canal and the perianal skin. The fistula may develop after an anorectal abscess has been drained operatively, or the fistula may develop spontaneously; less commonly, inflammatory bowel disease may cause a fistula-in-ano.
- B. The fistula is characterized by persistent purulent or feculent drainage, soiling the underwear.
- C. The fistula orifice can be seen peripheral to the anal verge. Complex fistulae may have multiple tracts with multiple orifices ("peppermint anus").

II. Treatment of Fistula-in-Ano

- A. Fistulae will not resolve without definitive treatment. The more common type of fistula, located at the anorectal junction, has an external opening where it can be drained operatively. The entire epithelialized tract must be found and obliterated.
- B. Goodsall's rule predicts the course of simple fistulae that exit the skin within 3 cm of the anal verge. Anterior fistulae go straight toward the anorectal junction; posterior fistulae follow a curving course toward the posterior midline and enter the anorectal junction.
- C. Occasionally, it can be difficult to distinguish a pilonidal cyst-sinus (coccygeal region) from a fistula-in-ano. Probing the tract under anesthesia usually will reveal its origin.
- D. **Fistulotomy:** For fistulae that do not cross both internal and external sphincters, the tract should be unroofed and curetted. The lesion should then be allowed to heal by secondary intention.
- E. **Fistulectomy:** Deeper fistulae should be treated by coring out the epithelialized tract to its origin; however, the fistula may recur.
- F. **Seton Procedure:** Complex fistulae or fistulae that traverse the sphincter can be treated by looping a heavy suture through the entire tract under tension. The suture should be tightened weekly until it "cuts" gradually to the surface, the tract will heal gradually behind the suture (analogous to a wire cutting through a block of ice - the ice refreezes after the wire passes).
- G. **Palliative Colostomy:** Extremely severe fistulization throughout the perineum may require fecal diversion for symptomatic relief.

Colorectal Cancer

John Craig Collins, M.D.

I. Clinical Evaluation of Colorectal Cancer

- A. Colorectal cancer is the second most common solid malignancy in adults and the second leading cause of cancer death in the U.S.
- B. Flexible sigmoidoscopy for screening is indicated for asymptomatic,

healthy adults over age 50. All adults with anemia or guaiac positive stools must be evaluated for colorectal cancer in the absence of other causes; older adults (>40) should be evaluated even if other sources of bleeding have been found. Hemorrhoids and cancer can coexist.

- C. Flexible sigmoidoscopy plus air contrast barium enema is adequate to evaluate the colon when the source of bleeding is thought to be benign anorectal disease.
- D. Total colonoscopy should be performed for any adult with gross or occult rectal bleeding and no apparent anorectal source.
- E. Left sided or rectal lesions are characterized by blood streaked stools, change in caliber or consistency of stools, obstipation, alternating diarrhea and constipation, tenesmus (involuntary sphincter spasm during defecation).
- F. Right sided lesions are characterized by a triad of iron deficiency anemia, melena, a right lower quadrant mass, and weakness.
- G. Cancers occasionally present as a large bowel obstruction, perforation or abscess.

II. Laboratory Evaluation

- A. CBC with indices (hypochromic, microcytic anemia). Liver function tests may sometimes be elevated in metastatic disease.
- B. **Carcinoembryonic antigen (CEA):** Carcinoembryonic antigen is not a screening test; may be elevated in colorectal cancer but it is a nonspecific test (also elevated in other malignancies, some inflammatory bowel disease, cigarette smokers, and some normal persons). Valuable in monitoring the response to treatment and as a marker for recurrence or metastases requiring adjuvant therapy. Should be obtained prior to resection of the tumor and at intervals postoperatively.
- C. Colorectal cancer is detected by total colonoscopy with biopsies. Barium enema may complement colonoscopy since BE shows the exact anatomic location of the tumor. A chest X-ray should be done to search for metastases to the lungs.

III. Management of Colorectal Carcinoma

- A. Surgical resection is indicated for colorectal adenocarcinoma, regardless of stage. Resection of the primary lesion prevents obstruction or perforation.
- B. Extremely advanced rectal lesions, which are not resectable, may be candidates for palliative radiation and a diverting colostomy.
- C. The extent of resection is determined by the relationship of the lesion to the lymphatic drainage and blood supply of the colon.
 - 1. **Cecal or Right Colon:** Right hemicolectomy.
 - 2. **Hepatic Flexure:** Extended right hemicolectomy.
 - 3. **Mid-transverse Colon:** Transverse colectomy or extended left or right hemicolectomy.
 - 4. **Splenic Flexure or Left Colon:** Left hemicolectomy.
 - 5. **Sigmoid Colon:** Sigmoid colectomy.
 - 6. **Upper or Middle Rectum:** Low anterior rectosigmoid resection with primary anastomosis.
 - 7. **Lower Rectum:** Abdominoperineal resection with permanent end colostomy, or local excision in selected cases.

D. Preoperative Bowel Preparation

1. Mechanical cleansing of the lumen, followed by decontamination with nonabsorbable oral antibiotics decreases the chance of infectious complications and allows for primary anastomosis.
 2. Partially obstructed patients should be prepped with caution because of the risk of perforation. Fully obstructed patients cannot be prepped and must have a temporary colostomy.
 3. Polyethylene glycol solution (GoLyte) is usually administered as 1 liter over 12 hours on the day before surgery. Nichols-Condon prep consists of 1 g neomycin sulfate and 1 g erythromycin base P.O. at 2:00, 3:00 and 11:00 pm the day before operation. Cefotetan 1 gm IV 30 mins. preop.
- E. Adjuvant chemotherapy based on 5-fluorouracil and levamisole for advanced colon lesions with the addition of pelvic radiation for advanced rectal tumors. Adjuvant therapy is reserved for locally advanced lesions (B2) or those with metastases to regional lymph nodes or distant organs (C1, C2, D).
- F. Staging workup (CT of chest, abdomen and pelvis) usually is done postoperatively since it is unnecessary for very early lesions, and it does not change operative management. Pathologic staging of the tumor is done postoperatively by histologic examination of the surgical specimen.

Staging of Colorectal Carcinoma

I. Dukes' Classification

Stage A: Confined to the bowel wall.

Stage B: Extends through the bowel wall; lymph nodes negative.

Stage C: Metastatic to regional nodes.

II. Astler-Coller Modification of Dukes' Classification (Most commonly used classification)

Stage A: Limited to mucosa and submucosa. Nodes negative.

Stage B1: Extends into but not through muscularis propria; nodes negative.

Stage B2: Extends through muscularis propria; nodes negative.

Stage C1: Same as B1, except nodes positive.

Stage C2: Same as B2, except nodes positive.

III. TNM Classification (International standard)

TX: Tumor cannot be assessed.

T0: No tumor in specimen (prior polypectomy done).

Tis: Carcinoma in situ.

T1: Invades into submucosa.

T2: Invades into muscularis propria.

T3: Invades through muscularis propria.

T4: Invades adjacent organs or into free peritoneal cavity.

NX: Nodes cannot be assessed.

N0: No regional nodal metastasis.

N1: 1-3 nodes positive.

N2: More than 3 nodes positive.

N3: Central nodes positive.

MX: Presence of distant metastases cannot be assessed.

M0: No distant metastasis.

M1: Distant metastasis present.

IV. Management of Obstructing Carcinomas of the Left Colon

- A. Correct fluid deficits and electrolyte abnormalities. Nasogastric suction is useful, but it is not adequate to decompress the acutely obstructed colon.
- B. The Hartmann procedure is indicated for distal descending and sigmoid colon lesions. This procedure consists of resection of the obstructing cancer and formation of an end colostomy and blind rectal pouch. The Colostomy can be taken down and anastomosed to the rectal pouch electively.
- C. Primary resection with temporary end colostomy and mucous fistula should be done for lesions of transverse and proximal descending colon. This procedure consists of resection of the obstructing cancer and creation of a functioning end colostomy and a defunctionalized distal limb with separate stomas. The colostomy can be taken down, and continuity can be restored electively.
- D. An emergency decompressive loop colostomy can be considered for acutely ill patients. A hemicolectomy can be completed after four to six weeks.
- E. A primary anastomosis may be done in selected patients with a prepared bowel.

V. Management of Obstructing Carcinomas of the Ascending Colon

- A. Correct fluid deficits, and electrolyte abnormalities, and initiate nasogastric suction.
- B. A right hemicolectomy with primary anastomosis of the terminal ileum to the transverse colon can be performed on most patients. A temporary ileostomy is rarely needed.

Diverticulitis

I. Pathogenesis

- A. By age 50 one third of the patients have diverticulosis coli, and approximately two thirds have diverticulitis by age 80. 10-20% of patients with diverticulosis will have complications of diverticulitis or diverticular hemorrhage.
- B. **Causes of Diverticulosis:** Aging, elevation of colonic intraluminal pressure, and decreased dietary fiber. Diverticuli occur where nutrient arteries penetrate the muscularis propria. Eighty-five percent are found in the sigmoid colon.

II. Clinical Presentation of Diverticulitis

- A. Usually >60 years of age; abrupt onset of unremitting left-lower quadrant abdominal pain, fever, alteration in bowel pattern. Diverticulitis of the transverse colon may simulate ulcer pain; diverticulitis of the cecum and redundant sigmoid may resemble appendicitis. Right sided diverticulosis is more common among Asians (>75%) than among Europeans.

- B.** Frank rectal bleeding is usually not seen with diverticulitis.
- C. Physical Exam:** Left-lower quadrant tenderness. Abdominal examination is often deceptively unremarkable in the elderly and in persons taking corticosteroids. Leukocytosis may occur.
- D. Differential Diagnosis:**

Elderly: Ischemic colitis Carcinoma Volvulus Colonic Obstruction Penetrating ulcer Nephrolithiasis/urosepsis	Middle Aged and Young: Appendicitis Salpingitis Inflammatory bowel disease Penetrating ulcer Urosepsis
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III. Diagnostic Evaluation

- A. Plain X-rays** may show ileus, obstruction, mass effect, ischemia, perforation.
- B. CT scan** is the test of choice to evaluate acute diverticulitis; used for staging the degree of complications and ruling out other diseases.
- C. Contrast Enema:** Water soluble contrast is safe, and useful in mild-to-moderate cases of diverticulitis when the diagnosis is in doubt.
- D. Endoscopy:** Acute diverticulitis is a relative contraindication--exclude perforation first. Used when the diagnosis is in doubt to exclude the possibility of ischemic bowel, Crohn's disease, or carcinoma.
- E. Ultrasound:** May be a helpful to evaluate acute diverticulitis. Intestinal gas often interferes with the exam.
- F. Complete Blood Count:** May show leukocytosis

IV. Treatment

A. Outpatient Treatment:

- 1. **Clear liquid diet**
- 2. **Oral antibiotics:**
 Ciprofloxacin (Cipro) 250-500 mg PO bid or 200-300 mg IV q12h
AND
 Metronidazole (Flagyl), 500 mg PO 2 times a day.

B. Inpatient Treatment:

- 1. Severe cases should be hospitalized for gastrointestinal tract rest (NPO), intravenous fluid hydration, correction of electrolyte abnormalities, and antibiotics.
- 2. Nasogastric suction is initiated if the patient is vomiting or if there is abdominal distention.
- 3. Antibiotic coverage for enteric gram-negative and anaerobic organisms
 Ampicillin 1-2 gm IV q4-6h **AND**
 Gentamicin or tobramycin 100-120 mg IV (1.5-2 mg/kg), then 80 mg IV tid (5 mg/kg/d) **AND**
 Cefoxitin (Mefoxin) 2 g IV q8h **OR**
 Metronidazole (Flagyl) 500 mg q6-8h (15-30 mg/kg/d) or Clindamycin (Cleocin) 600-900 mg IV q8h for more severe disease.
 Monotherapy with second-generation cephalosporin (eg, cefoxitin, cefotetan) or extended-spectrum penicillins (eg, piperacillin-tazobactam, ampicillin-sulbactam) may be used.

- C.** Frequently reassess the abdomen for the first 48-72 hours.

Improvement should occur over 48-72 hours with decreased fever, leukocytosis, and abdominal pain. Failure to improve or deterioration are indications for reevaluation and consideration of surgery. Analgesics are avoided because they may mask acute deterioration, and they may obscure the need for urgent operation.

- D. Oral antibiotics should be continued for 1-2 weeks after resolution of the acute attack. Ciprofloxacin, 500 mg, PO bid.
- E. After the acute attack has resolved, clear liquids should be initiated, followed by a low residue diet for 1-2 weeks. Then change to a high-fiber diet with psyllium.

V. Complications of Diverticulitis

A. Fistula:

- 1. With repeated attacks of diverticulitis, a fistulous tract can form between bowel, urinary bladder, integument, pelvic floor, or vagina.
- 2. Colovesicular fistula is the most common. Pneumaturia and recurrent urinary tract infections, characterized by multiple organisms may occur. Reflux of contrast into the urinary bladder during contrast enema confirms the diagnosis.
- 3. A segmental colon resection is usually performed after the acute diverticulitis has resolved.

B. Intestinal Obstruction: After repeated episodes of diverticulitis, the colon becomes fixed, fibrotic, stenosed, and obstruction may result. Surgical resection is indicated.

C. Abscess:

- 1. If no response to medical therapy within 24-48 hours, or if an abdominal mass is palpable, consider the possibility of an intra-abdominal abscess, and obtain an abdominal CT scan.
- 2. Obtain early surgical consultation for percutaneous drainage followed by surgical drainage.

D. Perforation and Peritonitis:

- 1. Diverticular perforation will result in peritonitis.
- 2. Pain becomes severe, with peritoneal signs (guarding, rebound tenderness, rigidity), fever, tachycardia, elevation of white blood count. Most patients are toxic and require prompt surgical intervention.
- 3. Perforation is indicated by free peritoneal air that may be visible below diaphragm on a chest x-ray. Perforated viscus from an ulcer is a much more common cause of free air.

VI. Surgical Therapy

- A. An emergency sigmoid colectomy with proximal colostomy is indicated for attacks of diverticulitis associated with sepsis, peritonitis, obstruction, or perforation.
- B. Elective sigmoid resection is indicated for second or subsequent attacks of diverticulitis, or for attacks with complications managed nonoperatively (eg, percutaneous CT-guided drainage of an abscess), or if carcinoma, or a single episode in a young patient (<40-50 years old), or an immunosuppressed patient.

C. Operative Procedures

- 1. **Single Stage Procedure:** This procedure is usually performed as an elective procedure after resolution of the acute attack of diverticulitis.

The segment containing inflamed diverticulum (usually sigmoid colon) is resected with primary anastomosis. A bowel prep is required.

- 2. Two Stage Procedure:** This procedure is indicated for acute diverticulitis with obstruction or perforation with an unprepared bowel. The first stage consists of resection of the involved segment of colon, with end colostomy and either a mucous fistula or a Hartmann rectal pouch. The second stage consists of a colostomy take-down and reanastomosis after 2-3 months.
- 3. Percutaneous Drainage of Diverticular Abscess:** CT guided drainage of accessible, well-circumscribed abscess cavities can be performed. A one-stage resection is done later as an elective procedure.

Mesenteric Ischemia and Infarction

Russell A. Williams, M.D.

I. History

- A. Acute:** Classic history of pain out of proportion to physical findings. Severe diffuse mid-abdominal pain (abrupt onset), bloody diarrhea, vomiting.
- B. Chronic:** Colicky postprandial epigastric pain (mesenteric angina) often occurring 30-60 minutes after meals. Aversion to food, weight loss.
- C.** History often includes peripheral vascular disease (claudication), coronary artery disease, atrial fibrillation, stroke, hyperlipidemia, diabetes.

II. Physical Exam

- A. General:** Moderate to severe distress occurs in acute cases with cachexia.
- B. Vitals:** Tachycardia, low-grade temperature elevation, orthostatic hypotension.
- C. Heart:** Irregularly irregular rhythm (suggests emboli from atrial fibrillation).
- D. Abdomen:** Diminished bowel sounds, abdominal bruits; often findings of tenderness or distention are minimal. Pulsatile supraumbilical mass suggests aortic aneurysm.
- E. Rectal:** Occult or gross blood.
- F. Extremities:** Diminished or absent pulses; bruits, loss of hair on legs, arterial ulcers, embolic gangrene of toes ("trash foot").
- G.** Chronic mesenteric ischemia is associated with systemic vascular disease and weight loss.

III. Laboratory Evaluation

- A.** Electrolytes (prerenal azotemia, metabolic acidosis). CBC (leukocytosis).
- B.** Abdominal series ("thumbprinting" suggests thickening of bowel wall; pneumatosis intestinalis portal vein gas).
- C.** Mesenteric angiography confirms or excludes the diagnosis.

IV. Differential Diagnosis: Peritonitis (secondary to acute appendicitis),

acute cholecystitis, perforated viscus, peptic ulcer, gastroenteritis, pancreatitis, bowel obstruction, carcinoma, ruptured aortic aneurysm.

V. Surgical Therapy of Mesenteric Infarction

- A.** Acute mesenteric infarction is a surgical emergency. Main options include superior mesenteric artery thrombectomy, aortomesenteric bypass graft; superior mesenteric endarterectomy with patch angioplasty.
- B.** A planned "second look" laparotomy may be used to confirm viability of bowel 24-48 hours after the initial revascularization.

Intestinal Obstruction

I. History

- A.** Crampy abdominal pain, vomiting, obstipation. History of previous laparotomy, hernia, colon cancer, gynecologic malignancy, cholelithiasis, chronic constipation, abdominal trauma; use of opiates, anticholinergics.
- B.** Feculent emesis and distention suggests distal obstruction. Pain is often colicky at first; progressing to generalized, diffuse abdominal pain. Initially crampy or colicky pain with exacerbations at intervals of 5-10 minutes. Pain later becomes diffuse with fever. Localizes to periumbilical region in small bowel obstruction; localizes to lower abdomen in large bowel obstruction.
- C.** Extremely severe pain suggests closed loop obstruction with intestinal ischemia.
- D.** Immobilization, weight loss, recent pelvic or abdominal operation, electrolyte abnormalities, and some medications may predispose to adynamic small bowel ileus or colonic pseudo-obstruction (Ogilvie's syndrome).

II. Physical Exam

- A. General:** Moderate to severe distress.
- B. Vitals:** Tachycardia, orthostatic hypotension, fever.
- C. Skin:** Cool, pale, clammy.
- D. HEENT:** Fetid, foul breath (often recognizable from across the room).
- E. Abdomen:** Healed laparotomy incisions, hernias (incisional, umbilical, epigastric, inguinal, femoral, obturator). Bowel sounds are initially increased with rushes and borborygmi; later, bowel sounds become decreased or silent. Distention, tympany to percussion. Diffuse rebound tenderness suggest peritonitis secondary to perforation.
- F. Rectal:** The rectal vault may be empty or impacted with feces.

III. Laboratory Evaluation

- A.** CBC (leukocytosis with left shift); electrolytes (hypokalemic, hyponatremic metabolic alkalosis from vomiting, or lactic acidosis from infarcted bowel).
- B.** Amylase will be elevated in pancreatitis and bowel perforation.
- C. Abdominal Films:** Dilated loops of small or large bowel with air/fluid levels and a "stairstep" appearance.
- D.** Water-soluble contrast enema: Demonstrates large bowel obstruction or

volvulus; the enema may resolve fecal impaction by inducing an osmotic diarrhea.

IV. Differential Diagnosis of Intestinal Obstruction

- A. Causes of Small Bowel Obstruction:** Adhesions (previous laparotomy), hernias, inflammatory strictures, superior mesenteric artery syndrome, gallstone ileus, adynamic ileus, malnutrition, internal hernia.
- B. Causes of Large Bowel Obstruction:** Colon cancer, sigmoid or cecal volvulus, diverticulitis, fecal impaction, adynamic ileus, pseudo-obstruction (Ogilvie's syndrome).

V. Treatment of Intestinal Obstruction

- A.** Restore normal intravascular volume with fluid resuscitation. Place a Foley catheter and maintain urine output 0.5-1 mL/hr. Correct hypokalemia and hypomagnesemia.
- B.** Initiate nasogastric suction (Levine tube to low intermittent suction). Replace the nasogastric output with an equal volume of 0.5 NS with 20-40 mEq/L potassium every 8 hours.

C. Small Bowel Obstruction

- 1. Nonoperative treatment with rehydration and nasogastric decompression is appropriate for partial obstructions thought to be due to adhesions. If such patients improve, continued nonoperative treatment is justified.
- 2. Immediate operation is mandatory for obstructions due to incarcerated hernias, and surgery should be strongly considered for all advanced or total obstructions.

D. Large Bowel Obstruction

- 1. A rapid workup should be performed to distinguish among impactions, pseudo-obstructions, and true obstructions of the colon. Manual disimpaction followed by hyperosmolar radiographic contrast enema is therapeutic for fecal impaction.
- 2. Complete large bowel obstruction due to colon cancer, diverticulitis or volvulus require immediate laparotomy with resection of the obstructed segment. Primary anastomosis may be performed for right-sided lesions. Left sided colonic obstructions require a diverting end-colostomy and Hartmann pouch or mucous fistula.
- 3. Colonoscopic decompression is standard treatment for Ogilvie's syndrome, and may permit nonoperative detorsion of a sigmoid volvulus.

Acute Pancreatitis

Russell A. Williams, M.D.

I. Diagnosis of Acute Pancreatitis

- A. Pancreatitis usually presents as abdominal pain associated with elevated pancreatic enzymes. Pain is typically epigastric or in the left upper quadrant, and the pain is described as constant, dull, or boring; radiation of the pain to the mid-back and worsening in the supine position may occur.
- B. Low-grade fever to 101°F is common. Higher temperature may indicate infectious complications. Acute pancreatitis may present with volume depletion, manifesting as hypotension or shock, due to vomiting, hemorrhage, or third spacing of fluid into the retroperitoneum.
- C. Patients may have a distended abdomen, and bowel sounds may be absent. Epigastric tenderness and localized rebound may be elicited.
- D. Bleeding into the pancreatic bed may rarely manifest as ecchymoses of the flanks (Grey Turner's Sign) or as periumbilical bleeding (Cullen's sign).

II. Etiology: Identification of the etiology of an attack of pancreatitis is essential to prevent recurrences.

A. Alcohol: Alcoholic pancreatitis can develop after an alcohol binge.

B. Gallstones:

- 1. Ultrasonography is the procedure of choice to visualize gallstones. 65-75% of patients with "idiopathic" acute pancreatitis have occult gallstones or biliary sludge.

2. Conditions that Predispose to Biliary Stones: Prolonged fasting (total parenteral nutrition, dieting), pregnancy.

C. Hypertriglyceridemia: Pancreatitis is associated with triglyceride levels >1000 mg/dL; lipid-reducing therapy will prevent recurrences.

D. Abdominal Trauma: Trauma such as an automobile accident can result in acute pancreatitis.

E. Postoperative: Pancreatitis may occur after upper abdominal, renal, or cardiovascular surgery.

F. Hypercalcemia: Pancreatitis has been reported with hypercalcemia.

G. Pregnancy: Acute pancreatitis is most likely during the third trimester and in the 6 weeks postpartum; it is usually related to alcohol abuse or gallstone disease.

H. Anatomic Causes of Pancreatitis: Duodenal diverticula, choledochoceles, pancreatic or ampullary strictures, pancreas divisum, tumors of the pancreas.

I. Infections: Viruses, parasites, and bacteria may cause pancreatitis.

J. Vasculitis: Pancreatitis may be an initial manifestation of systemic vasculitis.

K. Drugs: Nonsteroidal anti-inflammatory drugs, erythromycin, thiazides, dideoxyinosine (ddI), pentamidine, sulfonamides, and 5-aminosalicylate (5-ASA).

L. Other Causes: Acute pancreatitis may occur as a complication of endoscopic retrograde cholangiopancreatography, hereditary pancreatitis, scorpion stings (found in Trinidad), organophosphate insecticides, and other toxins.

III. Laboratory Evaluation

- A. Elevated amylase is not pathognomonic for pancreatitis. Ruptured ectopic pregnancy, tubo-ovarian abscess, ovarian cysts, duodenal perforation, and mesenteric infarction may result in moderate hyperamylasemia. Clearance of amylase is reduced in renal failure, resulting in up to a threefold elevation.
- B. In acute pancreatitis the amylase elevation is generally more pronounced than in other settings; values are usually at least 3 times normal. Mild hyperamylasemia may be seen in asymptomatic alcoholics and in acute cholecystitis or cholangitis.
- C. An elevated WBC count is common in pancreatitis.
- D. **Isoamylase Determination:** Distinguishes pancreatic amylase from salivary amylase. Elevation of salivary isoamylase occurs with mumps, pneumonia, lung tumors, and breast or prostate cancers.
- E. **Macroamylasemia**
 - 1. An elevation of serum amylase may result from low renal excretion of amylase, and is not an unusual finding in the normal population.
 - 2. Macroamylasemia occurs in 1% of all healthy persons and in 2.5% of patients with hyperamylasemia. Diagnosis is through detection of a low amylase clearance ratio (ACR).
$$\text{ACR} = \frac{\text{Urine Amylase} \times \text{Serum Creatinine}}{\text{Serum Amylase} \times \text{Urine Creatinine}} \times 100$$

With macroamylasemia, the ACR is less than 0.2%.
 - 3. Patients with macroamylasemia and pancreatitis may be diagnosed on the basis of an elevated serum lipase activity.
- F. **Lipase**
 - 1. Lipase is more pancreas specific than amylase.
 - 2. Increases may be due to chemotherapy, radiotherapy, alcohol, bile induced enteritis, extrahepatic biliary obstruction; hepatic failure or necrosis; chronic active hepatitis; alcoholic cirrhosis. In these disorders, the lipase level seldom exceeds the upper limit of normal by more than 3 times.

IV. Imaging Studies

A. Radiographic Studies

- 1. Flat and upright films of the abdomen help exclude perforated viscus (free air under diaphragm).
- 2. **Nonspecific Findings of Acute Pancreatitis:** Adynamic ileus or a sentinel loop (localized jejunal ileus). Pancreatic calcifications may be found with chronic pancreatitis.

B. Ultrasonography

- 1. Useful for evaluation of the biliary tract for gallstones, increased gallbladder wall thickness, and pericholecystic fluid associated with cholecystitis, or bile duct dilation due to obstruction.
- 2. Acute pancreatitis is indicated by reduced pancreatic echogenicity, enlargement, or ductal dilation. The pancreas cannot be visualized in 40% due to overlying bowel gas.

C. Computed Tomography (CT) Scanning

- 1. Contrast-enhanced CT scans have a sensitivity of 90% and a specificity of 100% for the diagnosis of acute pancreatitis.
- 2. **Indications for CT Scan:** Acute pancreatitis in patients who are

seriously ill or if the diagnosis uncertain.

D. Endoscopic Retrograde Cholangiopancreatography (ERCP): Not routinely indicated during an attack of acute pancreatitis, but it may be useful in the following situations:

1. Preoperative evaluation of suspected traumatic pancreatitis.
2. Suspected biliary pancreatitis with severe disease that is not improving and may need sphincterotomy and stone extraction.
3. In patients older than 40 years with no identifiable cause, ERCP is indicated once the attack of pancreatitis has subsided to determine the etiology.

V. Assessment of Prognosis

A. Ranson's Criteria

1. Used to assess prognosis early in the course of acute pancreatitis.
2. Overall, mortality from acute pancreatitis is approximately 1% in patients with less than 3 signs, 15% with 3 or 4 signs, 40% with 5 or 6 signs, and 100% with 7 or more signs.

Ranson's Criteria for Alcoholic Pancreatitis:

At Admission:

1. Age over 55 years
2. WBC $>16,000/\text{mm}^3$
3. Blood glucose $>200 \text{ mg/dL}$ (in a nondiabetic)
4. Serum LDH $>350 \text{ IU/L}$
5. AST $>250 \text{ U/L}$

During Initial 48 Hours:

1. Hematocrit drop $>10\%$ points
2. BUN rise $>5 \text{ mg/dL}$
3. Arterial $\text{PO}_2 <60 \text{ mm Hg}$
4. Base deficit $>4 \text{ mEq/L}$
5. Serum calcium $<8.0 \text{ mg/dL}$
6. Estimated fluid sequestration $>6 \text{ L}$

Ranson's Criteria for Nonalcoholic Pancreatitis:

Admission

1. Age over 70 years
2. WBC $>18,000/\text{mm}^3$
3. Blood glucose $>220 \text{ mg/dL}$ (in a nondiabetic)
4. Serum LDH $>400 \text{ IU/L}$
5. AST $>250 \text{ U/L}$

Initial 48 hours

1. Hematocrit drop $>10\%$ points
2. BUN rise $>2 \text{ mg/dL}$
3. Base deficit $>5 \text{ mEq/L}$
4. Serum calcium $<8.0 \text{ mg/dL}$
5. Estimated fluid sequestration $>4 \text{ L}$

VI. Complications of Pancreatitis

- A. Pseudocyst is a pancreatic fluid collection that may regress or progress to a mature pseudocyst. Pseudocysts are treated expectantly with monitoring for infection or growth associated with increasing symptoms. If pseudocyst does not resolve by 6 weeks or if complications occur, drainage is indicated either percutaneously or surgically.
- B. Necrotizing pancreatitis occurs after infection of the necrotic pancreatic tissue, often within 6 days after the episode begins.
- C. Systemic complications include shock, adult respiratory distress syndrome (ARDS), renal failure, and gastrointestinal bleeding.

VII. Medical Management of Pancreatitis

- A. **Supportive Medical Care for Local and Systemic Complications:** The majority of patients ($>80\%$) have rapid resolution of the inflammatory process and a noncomplicated course.
- B. **Replace Intravascular Volume:** Ringer's lactate, normal saline or colloids (albumin) are administered to restore hemodynamic stability and

to maintain a urine output of 0.5-1 cc/kg/h. Monitor urine output with a Foley catheter. Monitor central venous pressure and replace of calcium and magnesium deficits.

- C. Pancreatic Rest:** Oral feeding is withheld until clinical symptoms of nausea, vomiting, and abdominal pain have subsided. Total parenteral nutrition is usually necessary. After acute symptoms have resolved, feeding may be started, with gradual progression from liquids to a regular diet.
- D. Nasogastric Suction** is not indicated routinely, and should be used only if severe nausea, vomiting or ileus.
- E. Antibiotics:** Prophylactic antibiotics are necessary only in patients with a temperature greater than 101.5°F, suspected cholangitis, or severe pancreatitis. Cefoxitin (Mefoxin) 1-2 gm IV q6-8h.
- F. Analgesics:** Meperidine (Demerol) should be used because morphine may cause spasm of the sphincter of Oddi.

G. Medical Management

Somatostatin 250 mcg IV bolus, followed by 250 mcg/h x 48 hours **OR** Octreotide (Sandostatin) 200 mcg SC three times per day.

Ranitidine (Zantac) 50 mg IV q8h **OR**

Cimetidine (Tagamet) 300 mg IV q8h.

Cefotetan 1-2 gm IV q12h

Insulin may be needed if there is severe hyperglycemia.

Alcohol withdrawal prophylaxis may be required with chlordiazepoxide, thiamine 100 mg IM qd x 3d; folic acid 1 mg IM qd x 3d; multivitamin qd.

VIII. Surgical Management of Pancreatitis

- A. Indications for Surgical Management:** Exclusion of other intra-abdominal processes, abscess, infected, necrotizing pancreatitis or pseudocyst.
- B.** Operative management should be guided by CT imaging.
- C.** A cholecystectomy is indicated for biliary pancreatitis after an acute attack. The procedure is usually performed early, usually on hospital day 3 or 4 when amylase has returned to normal. Surgery may be performed during the acute attack if deterioration occurs. Endoscopic sphincterotomy is useful to remove obstructing stones in the sphincter of Oddi.
- D.** Severe necrotizing pancreatitis requires necrosectomy to remove necrotic pancreatic tissue and septic material. This is followed by local lavage and placement of large sump drains in the region of the pancreas, in the subphrenic, and in the subhepatic spaces.

IX. Pancreatic Pseudocyst

- A.** Clinical signs of pseudocyst include continuing abdominal pain, vomiting, nausea, epigastric tenderness, abdominal mass, and hyperamylasemia. CT and ultrasound are diagnostic.
- B. Pseudocyst Management**
 - 1. Pseudocysts are managed expectantly for 6-8 weeks until a mature cyst wall develops.
 - 2. Pseudocysts that are less than 5 cm in diameter usually resolve spontaneously.
 - 3. After a mature cyst wall has developed, internal drainage by cystogastrostomy, cystojejunostomy, or cystoduodenostomy is

completed.

4. A malignant pseudocyst should be excluded by biopsy of cyst wall.
5. Percutaneous CT guided drainage of pseudocysts may be considered after thickening of cyst wall has occurred.

Cholecystitis and Cholelithiasis

I. Clinical Evaluation

- A. History:** Acute cholecystitis is characterized by biliary colic, constant, right upper quadrant pain, occurring 30-90 minutes after meals, lasting several hours. The pain may extend to the epigastrium and radiate to back or right scapula. Pain has an abrupt onset with gradual relief. Pain is low grade fever 99-101 °F. Nausea, vomiting, anorexia.
- B.** Fatty food intolerance, dark urine, clay colored stools; bloating, jaundice, flatulence, obesity. Asian patients have an increased incidence of pigment stones.
- C.** History of gallstones, alcohol. History of fasting; high calorie diets, estrogen, diabetes.
- D.** Charcot's triad: right upper quadrant pain, jaundice, fever and chills, indicates ascending cholangitis.

II. Physical Exam

- A. Vital Signs:** P (mild tachycardia), T (low-grade fever), R (shallow respirations), BP (hypotension).
- B. Abdomen:** Epigastric or right upper quadrant tenderness, Murphy's sign (increased tenderness and inspiratory arrest during deep palpation of RUQ); guarding; rigidity, rebound.

III. Laboratory Evaluation

- A.** Moderate leukocytosis; mild elevation of bilirubin, increased alkaline phosphatase, AST, amylase. (If amylase is greater than 1000 U, consider biliary pancreatitis).
- B. Radionuclide scan (HIDA):** Positive if gallbladder does not visualize within 4 hours and radioisotope is visible in the common bile duct. Useful if bilirubin is less than 9 mg %, but may give a false positive if patient has not eaten for more than 48 hours.
- C.** Ultrasound of right upper quadrant shows dilation and gallstones; bile duct wall thickening, liver abscess, gas in biliary tree.
- D. Plain Abdominal X-ray:** Enlarged gallbladder shadow; calcifications in gallbladder; air in gallbladder wall (emphysematous cholecystitis); imprint of distended gallbladder on duodenum.

IV. Differential Diagnosis:

Cholecystitis, cholangitis, peptic ulcer, pancreatitis, appendicitis, gastroesophageal reflux disease, hepatitis, nephrolithiasis, hepatic metastases, gonococcal perihepatitis, pneumonia, angina.

V. Complications:

Perforated gallbladder, cholangitis, cholecystenteric fistula, gallstone ileus (stone passes into intestinal lumen via a fistula, and causes obstruction).

VI. Treatment

- A.** Remove the inflamed gallbladder during the acute stage (open or laparoscopic cholecystectomy).
- B.** Initially conservative, nonoperative measures (antibiotics, analgesics); followed later by elective cholecystectomy in a few weeks or months, after acute inflammation has resolved.
- C. Conservative Therapy**
 - 1. Antibiotics, IV fluids, electrolyte replacement. Operative drainage, should be considered for toxic patients for decompression of biliary tree.
 - 2. **IV Fluids:** Give IV crystalloids to restore intravascular volume. NG tube (10-18 F) at low constant suction (if nausea or vomiting); no oral intake.
 - 3. **Special Medications**
 - Gentamicin, 1.5-2 mg/kg, then 2-5 mg/kg/d IV **AND**
 - Metronidazole (Flagyl) 1.0 gm (15 mg/kg) over 1h, then 500 mg (7.5 mg/kg) IV q6h **OR**
 - Ticarcillin/Clavulanate (Timentin) 3.1 g IV q4-6h **OR**
 - Piperacillin/tazobactam (Zosyn) 4.5 gm IV q6h.
 - 4. **Symptomatic Medications**
 - Meperidine 50-100 mg IM q4-6h prn pain
- D. Nonsurgical Gallstone Dissolution Therapy:** Chenodeoxycholic acid ursodeoxycholate. Indicated only if adequate gallbladder function,

cholesterol stones (radiolucent), <2 cm in diameter. Stones recur if medicine is discontinued, monitor LFT's. Cholecystectomy is the treatment of choice for symptomatic gallstones.

Management of the Cholecystectomy Patient

I. Evaluation Before Early Cholecystectomy

- A. Confirm diagnosis with ultrasound and HIDA scan. Administer parenteral antibiotics and volume resuscitation.
 - 1. E. coli is the most common pathogen after cholecystectomy. With common duct obstruction, other pathogens, including Klebsiella spp, enterococcus, Clostridia spp, also are significant.
 - 2. Recommended prophylaxis consists of cefazolin (however, enterococcus coverage should be added in the "septic-appearing patient".
- B. Do not delay surgery in extremely ill patients with a palpable gallbladder or if complications develop such as emphysematous cholecystitis.
- C. Cholecystectomy may be performed in good risk patients presenting <72 hours after onset of symptoms.
- D. If the patient improves, early nonoperative conservative treatment may be considered with elective cholecystectomy scheduled at a later date. If the patient deteriorates or fails to improve, surgery should be performed.

Laparoscopic Cholecystectomy Procedure

- I. **Advantages of Laparoscopic Surgery:** Usually less postoperative; pain; reduced recovery time; several small puncture wounds instead of a large surgical incision; early return to work.

II. Procedure

- A. Insert a Hasson cannula through infraumbilical incision, and distend the peritoneal cavity with gas.
- B. The gallbladder is removed by inserting cannulae, special probes and instruments into abdominal cavity under video visualization.
- C. An intraoperative cholangiogram may be performed during laparoscopy.

- III. **Contraindications to Laparoscopic Cholecystectomy:** Adhesions, extreme gallbladder scarring, severe acute inflammation, bleeding, and other technically difficult situations.

Open Cholecystectomy Procedure

1. Prior to surgery place a nasogastric tube to decompress the stomach. The most commonly used incision is the Kocher's right subcostal.
2. Place incision 4 cm below and parallel to the costal margin, and extend it from the midline to the anterior axillary line. Open the anterior rectus sheath with a knife in the line of the incision. Divide the rectus muscle, and open the peritoneum between forceps.
3. Systematically explore the peritoneal cavity and note the appearance of the hiatus, stomach, duodenum, liver, pancreas, intestines, and kidneys. Palpate the gallbladder from the ampulla towards the fundus; then palpate the common duct, noting any dilation or foreign bodies. Carefully palpate colon for neoplasms.
4. Grasp the gallbladder with a Rochester-Pean or Pennington clamp near the fundus. Hold forceps in one hand, and introduce the right hand over the right lobe of the liver, permitting the liver to descend. Divide any adhesions to the omentum, colon or duodenum, and place a pack over these structures, then retract the structures inferiorly with a broad-bladed Deaver's retractor.
5. Inspect the anatomy of the biliary tree by carefully dividing the peritoneum covering the anterior aspect of the cystic duct, and continue dissecting into the anterior layer of the lesser omentum overlying the common bile duct. Bluntly dissect with a blunt dissector (Kitner), exposing Charcot's triangle bounded by the cystic duct, common bile duct and inferior border of the liver. The cystic artery should be seen in this triangle. Carefully observe the arrangement of the duct system and arterial supply. Do not divide any structure until the anatomy has been identified, including the cystic duct, common bile duct and porta hepatitis structures.
6. Pass a ligature around the cystic duct with a right-angle clamp, and make a loose knot near the common duct. Partially divide the cystic duct below the infundibulum, and place a small polyethylene catheter, attached to a syringe filled with saline, into the cystic duct for 1-2 cm. Tighten the ligature holding the catheter in position.
7. Attach a second syringe containing contrast material to the catheter, and remove all instruments. Place a sterile sheet, and slowly inject 15 cc of diluted dye into the common duct. Evaluate an operative cholangiogram to detect stones and evaluate the duct system. Make an X-ray exposure, then reposition table and make an exposure. Uncover the wound and proceed while the X-rays are processed.
8. Palpate the lower end of common bile duct and pancreas. Insert the index finger of the left hand into the foramen of Winslow. Allow the index finger to lie posterior to the second part of the duodenum and the head of the pancreas, and examine the pancreas for abnormal thickening due to inflammation, or stony hardness due to tumor. Palpate the ampulla, checking for stones or tumor. Hold the forceps on the gallbladder in the left hand to produce tension, and clear the cystic artery of soft tissue with a pledget held in forceps. Follow the artery to the gallbladder, and clamp it with a right angle clamp. Divide and ligate the artery close to the edge of the gallbladder, using clips or 000 silk.

9. Reaffirm the junction of the cystic duct with the common bile, then completely divide the exposed cystic duct, leaving a stump of at least 5 mm.
10. Hold the two forceps on the gallbladder in the left hand to put tension on the tissues between the gall bladder and the liver. Incise the peritoneum anteriorly over the gallbladder with a scalpel. Elevate the peritoneum from the gallbladder, and separate the gallbladder gently with sharp and blunt dissection. Tissue strands containing vessels should be cauterized before division.
11. Inspect the gallbladder bed for bleeding which may be cauterized or ligated. Control any persistent oozing from the bed with a small pack of hemostatic gauze.
12. Irrigate the site with saline. Routine drainage is unnecessary. However, if there is excessive fluids, place a soft rubber Penrose drain or closed suction drain in the area of the dissection, and bring it out through a separate stab wound in the right upper quadrant if desired. Inspect the operative field, including the ligatures on the arteries and the cystic duct. Grasp the peritoneum with a series of Kocher's forceps in continuity with the posterior rectus sheath, and approximate it with continuous nonabsorbable suture.
13. Irrigate the wound with saline and ligate any bleeding vessels. Approximate the rectus fascia and fascia of the oblique muscles with interrupted, nonabsorbable sutures. Irrigate the subcutaneous space with saline and close skin with staples, or absorbable subcuticular sutures. Secure the drain with a suture, and identify it with a safety pin. Cover the wound with a dressing.

Breast Cancer

John A. Butler, M.D.

Evaluation of Breast Masses

I. Guidelines for Breast Cancer Screening

- A. Women 20 years and older:** Monthly breast self examination.
- B. Ages 40-49:** Annual clinical examinations by physician and screening mammography at 2-3 year intervals.
- C. Age 50 and older:** Clinical examination and mammography annually.
- D.** Women with a personal history of breast cancer, history of breast cancer in a mother or sister, or with other risk factors, require closer surveillance.

II. Clinical Evaluation of Breast Masses

- A.** A detailed history should determine the presence of associated pain (especially if cyclical), nipple discharge, duration of mass, change in size, and color of any nipple discharges.
- B.** Determine patient age and menopausal status. The incidence of breast cancer rises rapidly in the fifth decade. Breast cysts are common before menopause and vary in size with menses.
- C.** Determine results of and time since the last clinical breast examination and the last mammogram. Assess breast self-examination practices and previous breast masses and biopsies.

D. Risk Factors for Breast Cancer

- 1. Female sex
- 2. Age over 50 years old
- 3. Previous breast cancer
- 4. Family history, particularly in first degree, maternal, or premenopausal relatives.
- E.** 80% of women with breast cancer have no risk factors other than being women and over 50 years old.

F. Physical Examination

- 1. First examine the patient in the supine position with her arms up and behind her head. This flattens the breast tissue and compresses it for examination.
- 2. Then have the patient sit up with arms first at her side and then behind her head. This facilitates examination of the breast contours and allows visualization of nipple inversion or tethering.
- 3. Examine for dimpling, asymmetry, lumps, thickened areas, changes in shape or contour. Carefully palpate both breasts and the axillae. Compress nipples to identify any discharge. Make a drawing of any irregularities and masses.
- 4. The characteristics of a lump have limited diagnostic value. Very discrete, smooth nodules are more likely to be benign; tenderness is usually associated with benignity. If the physical characteristics of the mass suggest benignity, it is reasonable to monitor a nodule during one menstrual cycle.

5. Assess mass for singular or multiple components, mobility, tenderness, and cystic or solid qualities.

III. Differential Diagnosis by Age of the Patient

- A. <30 Years Old:** The common causes are fibroadenoma, papillomatosis, abscess (especially if lactating), or fat necrosis.
- B. 30-50 Years Old:** Consider fibrocystic mastopathy, cancer, fatty lobule, or cystosarcoma phylloides.
- C. If Older than 50:** Cancer is the primary diagnosis, followed by fibrocystic mastopathy, fat necrosis, and cyst.

IV. Diagnostic Approach to a Breast Mass

- The goal of breast cancer screening is early identification of dominant breast masses or mammographic abnormalities (masses or microcalcifications). A dominant breast mass is a palpable breast lesion that is distinct from the surrounding breast nodularity, persistent through at least 2-3 menstrual cycles (if the patient is pre-menopausal), and is relatively unchanging. Although they generally represent benign processes, it is essential that they be worked-up to exclude malignancy.
- A **mammogram** should be obtained because mammographic abnormalities, such as masses, irregularities, asymmetry, and new suspicious microcalcifications require biopsy for diagnosis.
- Once a dominant mass has been identified, an ultrasound of the breast should be obtained to determine if the mass is cystic or solid. If the lesion is cystic, the fluid can be removed and the cyst collapsed. If the mass is solid, some form of tissue biopsy is required.
- Cyst Aspiration:** If physical characteristics support the diagnosis of a cyst, needle aspiration should be done. Aspiration is not necessary if ultrasound has determined that the mass is a cyst.
 - Swab the area of the breast with Betadine, and inject 1% lidocaine into the overlying skin using a 27-30 gauge needle.
 - Insert a 22 gauge needle into the mass and evacuate the cyst.
 - Non-bloody fluid should not be sent for cytology. If grossly bloody, the fluid should be analyzed by cytology, and biopsy considered. A compression dressing is placed over the breast, and the patient reexamined in 3-4 weeks.
 - If the lesion is found to be solid or if no fluid is obtained, use the 22 gauge needle to aspirate tissue (fine needle aspiration cytology).
 - If non-bloody fluid was removed, the patient can then be observed for recurrence.
 - Recurrence of cyst, or residual mass after aspiration, or presence of bloody aspirate requires further evaluation by ultrasound and/or biopsy.
- Fine-Needle Aspiration Cytology**
 - Sensitivity 65-98%. False-positive results are rare, but false-negative rates may be as high as 22%. If fine-needle aspiration is negative, biopsy is required.
 - Use a 10- or 20-mL syringe with a 22 gauge needle. When the needle enters the mass, apply suction by retracting the plunger and advance the needle. Direct the needle to different areas of the mass, maintaining suction on the syringe.
 - Slowly release suction before the needle is withdrawn from the

mass., and place the contents of the needle onto glass slides or put the cells in a fixative solution for pathologic examination.

- F. If the clinical exam, mammography, and fine-needle aspiration are benign, biopsy is not usually necessary because 98% of breast cancers will be detected by these measures.
- G. Open, excisional biopsy is the definitive method of diagnosis if the mass remains suspicious after these tests have been completed.

V. Nonpalpable (occult) Breast Lesions Detected by Mammogram

A. Evaluate with Breast Ultrasound

1. **If Solid:** The lesion should be evaluated by stereotactic needle biopsy.
2. **If Cystic:** Cyst aspiration may be considered as above, or follow the mass with breast self exam, ultrasound, clinical exam, and mammography. If the lesion becomes solid, evaluate with stereotactic needle biopsy or aspiration cytology. If it remains cystic, continue follow-up examinations.

VI. Important Diagnostic Caveats

- A. A normal mammogram is meaningless in the presence of a mass, and a mammogram can not rule out cancer.
- B. Mammography will miss 15-20% of breast cancers found on clinical examination. False-negative mammograms have been found in up to 63% of women younger than age 45.
- C. The majority of mammographic abnormalities and especially breast masses are benign (approaching 90+ percent in some series).
- D. Assure a follow-up examination for all patients, even if there is absolute certainty that the mass is insignificant or benign.
- E. A breast mass in a pregnant patient warrants the same timely evaluation as in a non-pregnant patient.
- F. Routine use of mammography in women less than 35 years old is usually unrewarding and potentially misleading and dangerous. Dense, hormonally active breast tissue in this group makes interpretation difficult and can not reliably define malignancy.

Excisional Breast Biopsy Technique

1. When mammography demonstrates a lesion suggestive of malignancy, the patient should have an x-ray on the morning of the operation and wire probes placed in the lesion for localization.
2. Infiltrate the skin and underlying tissues with 1% lidocaine with epinephrine. For lesions located within 5 cm of the nipple, a periareolar incision may be used of no more than half the areola circumference, or use a curved incision located over the mass and parallel to the areola.
3. Incise the skin and subcutaneous fat, then palpate the lesion and push it into the incision, and excise the mass. Send the specimen with the wire probes in place, for radiography to ensure that the abnormality has been fully exercised.
4. Close the wound with a 4-0 chromic catgut suture for the subcutaneous tissues, and 4-0 subcuticular Dexon for the skin. If

there is significant oozing, place a small Penrose drain or closed suction drain.

Urology

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Benign Prostatic Hyperplasia

I. Pathophysiology

- A. The prostate normally and naturally grows larger as men age. After age 40, benign prostatic hypertrophy commonly develops. BPH affects half of men by age 60, and at least 80% by age 80. 40% of 80 year old men will have had a prostatectomy.
- B. Benign prostatic hypertrophy is a nonmalignant neoplasm of the prostate and is not a precursor or predisposing factor to prostate cancer.
- C. Benign prostatic hyperplasia causes the syndrome of prostatism--irritative and obstructive urinary symptoms. The natural course of prostatism usually is that of waxing and waning symptoms.
- D. BPH is the most common cause of obstructive or irritative voiding symptoms in elderly men; however, prostate carcinoma and urethral stricture should always be considered.

II. Pathophysiology

- A. Prostatic stromal tissue is controlled by the adrenergic nervous system. Norepinephrine acts on alpha-1 receptors that control 80% of the activity of prostatic smooth muscle cells, causing contraction of the stromal component of the prostate.
- B. Prostatic tissue is also under hormonal control. Testosterone is converted to dihydrotestosterone by the 5-alpha reductase enzyme located in the prostatic epithelial cells. Dihydrotestosterone then binds to a cellular hormone receptor.
- C. Many men with palpably enlarged prostates are not symptomatic while some patients with small prostates have significant voiding symptoms or present with urinary retention.
- D. Obstruction forces the bladder to generate higher pressures than normal to achieve micturition. Increased muscle mass in the bladder leads to reduced bladder elasticity and compliance, which manifest as a reduction in bladder capacity.
- E. After a period of time, if the obstruction is not relieved, bladder smooth muscle begins to be replaced by connective tissue, leading to bladder failure. This process produces the classic obstructive voiding symptoms of hesitancy, intermittency, decreased force of the stream, postvoid dribbling, and a feeling of incomplete bladder emptying.

III. Clinical Evaluation of Benign Prostatic Hypertrophy

- A. The workup for benign prostatic hypertrophy includes a history, a digital rectal examination, and a prostate-specific antigen (PSA) blood test in men older than 45 years.

Symptoms of Prostatism

Irritative Symptoms

Nocturia
Urinary frequency
Urinary urgency
Dysuria
Incontinence

Obstructive Symptoms

Decreased force of stream
Urinary hesitancy
Terminal dribbling
Double voiding
Straining to urinate

B. The history should include a American Urological Association (AUA) Symptom Score questionnaire. This questionnaire assigns a numerical score to the common symptoms associated with benign prostatic hypertrophy. The higher the score, the more severe the obstruction. The questionnaire can also be used to gauge response to treatment.

1. Sensation of incomplete emptying?
2. Urinate again in <2 hours after emptying bladder?
3. Stop and start while urinating?
4. Difficulty postponing urination?
5. Weak stream?
6. Pushing or straining?
7. Typically get up to urinate at night? (number of times, up to 5)

The first six questions are scored as: 0 = never, 1= less than 20% of the time; 2 = 20-50%; 3 = 50%; 4 = >50%; 5 = almost always.

The total score is scored as follows: Mild = 0-7; moderate = 8 -18; severe = 19-35

C. Prostate cancer is suggested by a prostate nodule or a hardened area of the prostate on the digital rectal examination; the PSA level is usually elevated.

D. A urinalysis should be obtained to exclude urinary tract infection.

E. Prostate Specific Antigen: The normal range is 0 to 4. A PSA level of 4 to 6, although elevated, is statistically most likely to represent benign prostatic hypertrophy. If the examination and PSA level are normal, prostate cancer can safely be excluded.

F. Differential Diagnosis

1. The symptoms of BPH are not specific for prostate cancer because symptomatic and asymptomatic older men have the same rate of prostate cancer. Therefore, the patient's symptoms alone should not affect the decision to screen for prostate cancer.
2. The most common nonobstructive cause of obstructive voiding symptoms is diabetes mellitus. Diabetes mellitus leads to peripheral neuropathy, including the nerves of the bladder. Patients lose the sensation and then, due to overdistension of the bladder, develop detrusor muscle failure, resulting in obstruction. Glucose will be present on the urinalysis.
3. Urinary tract infection can mimic the irritative symptoms of BPH; however, pyuria will be present on urinalysis.
4. Hematuria is usually caused by BPH in older men, but urologic referral for cystoscopy may be required to rule out bladder cancer.
5. **Drugs That May Exacerbate Prostatism:** Alcohol, caffeine, first-generation antihistamines (cold remedies), tricyclic antidepressants, and urinary anti-spasmodic agents.

G. Complications of Benign Prostatic Hypertrophy

1. **Urinary tract infection** is one of the most frequent complications; can be recurrent or cause sepsis.
2. **Severe obstruction and hydronephrosis** may cause uremia that is manifest by malaise and nausea.
3. **Urinary retention** (200 to 300 mL post-voided residual urine) may present as abdominal pain with suprapubic pressure or suprapubic fullness.

IV. Treatment of Benign Prostatic Hyperplasia

- A. Patients with moderate symptoms should begin medical treatment.
- B. Severe symptoms warrants initiation of medical treatment, but surgical intervention will usually be required.
- C. **Medical Therapy**
 1. Most patients with BPH can be treated based on symptoms, but if PSA or UA are abnormal or if a prostate nodule is detected, then a urological evaluation is warranted.
 2. **Alpha-1-adrenergic Receptor Blockers**
 - a. Alpha-1 blockers relax the stromal (smooth muscle) component of the prostate, decreasing bladder outlet obstruction.
 - b. Long-acting and alpha-1 specific blockers are preferable to short-acting ones because of improved patient compliance and fewer side effects.
 - c. Terazosin (Hytrin), 5-10 mg qd, and doxazosin (Cardura), 2-8 mg qd, are long-acting alpha-1-blockers; once a day dosing is convenient.
 - d. Prazosin (Minipress) is the most frequently used short-acting alpha-1-blocker. Its main advantage is that it is inexpensive; 1-2 mg bid.
 - e. Alpha-1 blockers improve urine flow by 30-59% and decrease postvoid residual volume and voiding frequency.
 - f. The main side effects of alpha-blockers are dizziness (10%) and postural hypotension (7%), especially after the first dose. However, these side effects are usually mild and are mostly seen in patients with a history of hypertension. Normotensive patients may experience fatigue (7%).
 - g. The incidence and severity of these side effects can be decreased by bedtime administration.
 - h. Alpha-blockers work immediately, but they do not prevent progression of future prostatic growth.
 - i. Men with hypertension and benign prostatic hypertrophy may be able to control both problems with only one medication.
 - j. Alpha blockers are more effective in relieving symptoms than finasteride, and the combination of alpha-blockers and finasteride is not more effective than alpha blockers alone.
 3. **Hormonal Manipulation**
 - a. **Finasteride (Proscar)** is an antiandrogen that normally does not cause impotence. Finasteride is a 5-alpha reductase inhibitor that blocks prostatic conversion of testosterone to dihydrotestosterone, resulting in shrinkage of the prostate.
 - (1) Dosage is 5 mg per day.

- (2) Finasteride requires about 3-6 months to produce results, and clinical improvement is modest in most men, with only 33% to 50% reporting substantial clinical improvement.
- (3) Finasteride is used primarily for men with significant cardiac disease who can not tolerate alpha-1 blockers.

V. Surgical Therapy

A. Transurethral Resection of the Prostate

1. TURP is the most effective way to relieve symptoms of prostatism. Twenty-five percent of appropriately aged men undergo TURP.
2. The average total cost of TURP including hospitalization is about \$12,000. Good resolution of urinary symptoms is achieved in 90%.
3. The total morbidity from TURP is about 18%; impotence occurs in 10%. Most men develop retrograde ejaculation and report a "change" in orgasm, 3% report chronic incontinence, 5% become infected, and 1% die.
4. Twenty-five percent require repeat TURP within 10 years.
5. **Definite indications for Surgery:** Urinary retention with secondary hydronephrosis, increased creatinine, overflow incontinence, and recurrent urinary tract infections
6. **Relative indications for Surgery:** Recurrent gross hematuria, severe symptoms (96% improve)
7. Other than symptoms, there is no way to identify men more likely to benefit from surgery.

Prostate Carcinoma

The average age at diagnosis of prostate cancer is 73 years. The age-adjusted death rate from prostate cancer has not changed appreciably in 35 years.

I. Clinical Evaluation

- A. The prevalence of prostate cancer is 30% in men over the age of 50. One in six men will be diagnosed with prostate cancer during their lifetimes.
- B. Patients with prostate cancer may have obstructive urinary symptoms similar to benign prostatic hypertrophy; some patients may have weight loss and bone pain. Some may have no symptoms, only an elevated prostate specific antigen found on routine screening.
- C. **Physical Exam:** Digital rectal exam is used to assess the nodule for extension beyond prostate edge, firmness, fixation, or induration.
- D. **Prostate Specific Antigen (PSA)**
 1. PSA is a glycoprotein produced by both benign and malignant prostatic epithelium.
 2. Each gram of prostate tissue increases serum PSA by about 0.3 ng/mL, but each gram of prostate cancer increases serum PSA by about 3.5 ng/mL.

Normal PSA	<4.0 ng/mL
Borderline PSA	4-10
Elevated PSA	>10

3. A normal PSA does not rule out prostate cancer at any stage. However, a level above 10 ng/mL (and especially above 20 ng/mL) almost certainly reflects malignancy. Serial PSA levels that are increasing, even if still <10 ng/mL, probably indicate tumor.
4. A PSA > 4.0 ng/mL detects 46% of subsequently diagnosed prostate cancers, with a specificity of 91%.
5. PSA levels between 2 and 3 ng/mL were associated with a 5-fold increased risk of developing prostate cancer.
6. There is no effect of rectal exam on PSA when the level is low. However, there are substantial increases when the initial PSA is >10 ng/mL. Instrumentation and prostatic massage cause greater increases in PSA.

E. Transrectal Ultrasonography

1. Transrectal ultrasound is more sensitive than digital rectal exam, but still misses about 30% of known cancers.
2. The main indication of ultrasound is to guide transrectal biopsy of palpated lesions.

F. Problems with Prostate Cancer Screening

1. About 33% of men ages 50-70 have pathologic evidence of prostate carcinoma. No currently available diagnostic test can find most of these tumors.
2. PSA screening has not been shown to reduce morbidity and mortality from prostate cancer, and screening is extremely expensive.
3. Based on current evidence, screening for prostate cancer is unlikely to improve life expectancy substantially for men ages 50 to 70.
4. Screening is actually associated with a reduction in expected quality-adjusted life years because of treatment-related morbidity and mortality.

II. Urological Evaluation of Suspected Prostate Cancer

- A.** The prostate nodule is evaluated by transrectal ultrasound and prostate needle biopsies. If no transrectal ultrasound lesion is identified, then random sextet biopsies should be performed.
- B. Indications for Biopsy:** (1) Abnormal digital rectal exam (discrete, firm nodule), (2) Elevated prostate-specific antigen (regardless of digital rectal exam).
- C. Metastatic Evaluation:** SMA 18, chest x-ray, intravenous pyelogram or ultrasound, and bone scan.

III. Staging of Prostate Carcinoma

A. Tumor Node Metastases Classification (TNM)

<u>Tumor stage</u>	<u>TNM</u>
Incidental finding (TURP)	
≤3 microscopic foci (≤5%)	T1a
>3 microscopic foci (>5%)	T1b
Increased PSA	T1c
 Clinical tumor, limited to prostate	
≤1.5 cm	T2a
>1.5 cm or >1 lobe	T2b
 Tumor beyond prostate	
Not in seminal vesicles	T3
Seminal vesicles	T3
Fixed to pelvis, or invades locally	T4
 Metastatic disease	
Lymph nodes	N
Distant	M

B. Histopathologic Grade

Well-differentiated, slight anaplasia	G1
Moderate well-differentiated, moderate anaplasia	G2
Poorly differentiated, marked anaplasia	G3

C. Combining grade and TNM yields final stage

Stage 0	=	T1a or T2a	and	G1
Stage I	=	T1a or T2a	and	G2
Stage II	=	T1b, T2b	and	Any G
Stage III	=	T3	and	Any G
Stage IV	=	T4 or N or M	and	Any G

IV. Therapy of Early Stage Prostate Cancer

- Stage 0 disease can be followed
 - Stage I and II disease are best managed surgically with radical prostatectomy (with radiation therapy another option). Radical prostatectomy consists of removal of the prostate and seminal vesicles, and a staging pelvic lymph node dissection.
 - Stage III disease is managed with radiotherapy
 - Stage IV disease is managed with endocrine manipulation.
 - There is no firm evidence that any of these therapies is better than any other: therefore, "watchful waiting" is also a legitimate option.
 - Studies, particularly those from Scandinavia where watchful waiting is the standard of care, suggest that it is difficult to demonstrate a survival benefit (and especially a quality of life benefit) from most forms of therapy. Exceptions may be younger men (with a life expectancy of 10 years or more) with high grade but localized lesions (Stage I or II).
- #### G. Endocrine Therapy of Advanced Prostate Carcinoma
- Treatment of advanced malignancy (Stage IV) involves surgical or medical castration after development of symptoms (such as bone pain, obstructive symptoms, anemia) before instituting therapy.
 - Total blockade (e.g., leuprolide plus flutamide) may be slightly better than leuprolide alone.
 - Orchiectomy is an outpatient procedure that is the safest and least expensive option. There is no higher incidence of impotence with

orchiectomy than with medical castration therapies.

Renal Colic

Approximately 5% of the U.S. population will pass a urinary tract stone during their lifetime.

I. Pathophysiology

- A. Calcium-containing stones are the most common (70%).
- B. Magnesium-ammonium-phosphate stones, also known as struvite stones, are almost always associated with urinary tract infection by urea-splitting bacteria such as *Proteus mirabilis*. These are generally responsible for large staghorn calculi stones.
- C. Uric acid stones are less common and are radiolucent, making diagnosis by plain films alone difficult.
- D. Cystine stones are rare and associated with cystinuria, a rare autosomal recessive hereditary disorder.

II. Clinical Evaluation

- A. Renal colic is characterized as severe colicky pain that is intermittent, usually in the flank or lower abdomen. Patients usually can not find a "comfortable position," and the pain often radiates to the testes or groin. A history of previous stones, poor fluid intake, urinary tract infections, or hematuria is common.
- B. Obstruction located at the ureteropelvic junction causes pure flank pain, while upper ureteral obstruction causes flank pain that radiates to the testis. Midureteral stones cause lower abdominal pain and may mimic appendicitis or diverticulitis, but without localized point tenderness or guarding. Lower ureteral stones may cause irritative voiding symptoms and scrotal or labial pain.
- C. Patients with nephrolithiasis generally complain of nausea and vomiting. They commonly have gross or microscopic hematuria; fever and increased white blood cell count are also possible.
- D. Prior episodes of renal colic or a family history of renal stones is often reported.
- E. **Physical Examination**
 - 1. Generally the patient is agitated, diaphoretic, and unable to find a comfortable position.
 - 2. Hypertension and tachycardia are also common.
 - 3. Costovertebral angle tenderness is the classic physical finding; however, minimal abdominal tenderness without guarding, rebound, or rigidity may be present. Right or left lower quadrant tenderness or an enlarged kidney may sometimes be noted.

III. Differential Diagnosis: Appendicitis, salpingitis, diverticulitis, pyelonephritis, ovarian torsion, prostatitis, ectopic pregnancy, bowel obstruction, carcinoma.

IV. Laboratory Evaluation

- A. A urinalysis with microscopic, serum chemistries, BUN, creatinine,

complete blood count, and urine culture should be obtained. An elevated white blood cell count may occur, and when it is associated with fever, its presence usually indicates an associated infection. A significant number of white cells in the urine also suggests infection.

- B. A plain abdominal film may demonstrate a calcification along the course of the urinary tract, as 90% of all calculi are radiopaque. These films, however, are highly inaccurate, since calcifications may either be missed because of overlying bowel gas, or calcifications may be related to processes outside the urinary tract.

C. Intravenous Pyelogram

1. IVP is considered the gold standard, and it allows rapid assessment of the degree of obstruction, location of the stone and any renal function impairment.
 2. In cases of acute ureteral obstruction, the classic findings are a dense nephrogram with a delay in excretion of contrast. Visualization of the whole ureter above the stone (columnization) is sometimes demonstrated.
- D. **Ultrasound** has a limited role in the evaluation of acute renal colic, but it may be useful in patients with renal failure or an intravenous contrast allergy.

V. Management of Renal Calculi

- A. Most renal calculi will pass spontaneously, and only expectant management with hydration and analgesia is necessary.
- B. Obstruction associated with fever indicates urinary tract infection, and it requires prompt drainage with either a ureteral stent or percutaneous nephrostomy to prevent sepsis.

C. Indications for Admission

1. High fever, uncontrollable pain
2. Intractable nausea and vomiting with an inability to tolerate oral fluids
3. Solitary kidney

D. Inpatient Management

1. Vigorous intravenous hydration and intravenous antibiotics are important when infection is suspected.
 2. Parenteral narcotics are often necessary.
 3. Ketorolac (Toradol), an IM nonsteroidal drug, is effective and provides a good alternative to narcotics.
 4. Strain all urine in an attempt to retrieve spontaneously passed stones for X-ray crystallography analysis.
- E. Stones measuring 5 to 10 mm have a decreased likelihood of passage, and early elective intervention should be considered
- F. Extracorporeal shock-wave lithotripsy (ESWL) is the most common procedure for small renal or ureteral stones, and it requires no anesthesia. 80% of patients become stone-free after one treatment.
- G. Ureteroscopy with laser, ultrasound or electrohydraulic lithotripsy may be used for larger stones. Open surgical stone removal is rarely necessary.

H. Outpatient Management

1. Most patients with renal colic do not require admission.
2. Most stones measuring less than 4 mm will pass spontaneously (90-95%), and approximately 80% of these will pass within 4 weeks.

3. Patients should increase intake of oral fluids, take narcotic pain medication, and strain all urine.
 4. Close follow-up is necessary and plain abdominal films may be used to assess movement of the stone.
- I. Follow up Care**
1. After the stone has passed, a metabolic evaluation is important because 70% of patients will have a repeat stones if not diagnosed.
 2. Evaluation may include chemistry screening, calcium, uric acid, phosphorous, UA with micro, urine C&S, urine cystine (nitroprusside test), 24 hour urine collection for uric acid, calcium, creatinine. If increased serum calcium, check PTH level.

Urologic Emergencies

I. Acute Urinary Retention

- A.** Acute urinary retention is characterized by a sudden inability to void. It often presents with suprapubic pain and severe urgency. There is usually a history of preexisting obstructive voiding symptoms related to bladder outlet obstruction or poor detrusor function.
- B. Complications of Acute Urinary Retention**
1. Postobstructive diuresis
 2. Bladder mucosal hemorrhage
 3. Hypotension
 4. Sepsis
 5. Renal failure
 6. Autonomic bladder hyporeflexia
- C. Benign prostatic hyperplasia** is the most common cause of acute urinary retention in men over the age of 50.
1. Patients present with progressively worsening voiding difficulties, resulting in bladder overdistention and subsequent urinary retention.
 2. Prostate size on digital rectal examination has no bearing on the degree of outlet obstruction because minimal enlargement of the prostate can cause significant obstruction in some patients.
- D. Prostate cancer** accounts for 25% of patients with acute urinary retention. 10% of patients with prostate cancer initially present with bladder outlet obstruction.
- E. Additional Causes of Acute Urinary Retention:** Urethral strictures, bladder neck contractures, bladder stones, and acute bacterial prostatitis.
- F. Poor bladder function** can cause acute urinary retention. Common causes of poor bladder function include prolonged obstruction, diabetes mellitus, neurologic disorders (spinal cord injury, herniated vertebral disk, dementia), and medications.
- G. Urinary retention after surgery** sometimes temporarily develops in elderly men. There is usually preexisting bladder dysfunction or outlet obstruction that predisposes the patient to urinary retention.
- H. Anticholinergic medications** (antihistamines, antidiarrheals, antispasmodics, tricyclic antidepressants) can suppress bladder function. Sympathomimetic drugs (decongestants and diet pills) that

cause contraction of the smooth muscle in the bladder neck can precipitate an increase in outlet resistance.

II. Clinical Evaluation of Acute Urinary Retention

- A.** Patients will generally complain of an inability to void and suprapubic discomfort. A progressive history of difficulty voiding and irritative voiding symptoms, such as frequency, nocturia, or urgency is often noted.
- B.** Some patients are incontinent as a result of extreme overdistention of the bladder.
- C.** A past history of gonorrhea or trauma, that may have caused a stricture, should be sought, as well as the presence of underlying diseases and medications.
- D.** Palpate for a distended bladder and assess size and consistency of the prostate. Tenderness of the prostate on rectal examination suggests acute prostatitis; a diffusely hard or nodular prostate indicates carcinoma.
- E.** The penis should be examined to rule out phimosis, paraphimosis, or meatal stenosis.
- F.** A neurologic exam should be completed including anal sphincter reflex and perineal sensation.
- G. Laboratory Evaluation:** Serum electrolytes, blood urea nitrogen (BUN), creatinine, urinalysis, and culture.

III. Management of Acute Urinary Retention

- A.** Prompt drainage of the entire bladder contents with a Foley catheter is essential. Adequate volume replacement is necessary to prevent hypotension.
- B.** Lubrication with 2% lidocaine jelly (injected directly into the urethra with a syringe) will facilitate insertion of a urethral catheter.
- C.** Medium-sized catheters (#18 to #22 French) should be used because they tend to be stiffer and easier to insert than smaller ones, which can coil within the urethra.
- D.** In patients with large prostates, coude catheters, which have a curved tip, may be helpful. The curve of the coude catheter should be directed superiorly. Other methods of drainage include urethral sounds, filiforms with followers, and percutaneous suprapubic tubes.
- E.** Admission to the hospital is not required for most patients with acute urinary retention unless other associated factors, such as infection or renal failure, are present. Most patients can be managed with a Foley catheter and discharged home with oral antibiotics and a leg urine bag.

IV. Complications of Acute Urinary Retention

- A.** Postobstructive diuresis can occur; therefore, fluid and electrolyte balance should be monitored closely.
- B.** If significant post-drainage hemorrhage occurs, continuous bladder irrigation should be initiated.
- C.** Hypotension may result from either hypovolemia or a vasovagal reaction. However, it can be prevented by adequate volume replacement. Slow decompression of the bladder is not recommended.
- D.** Other complications of urinary retention include sepsis and renal failure due to longstanding obstruction.

V. Testicular Torsion

- A.** Testicular torsion is an emergency, and any delay in treatment may result in testicular loss. A four- to six-hour delay may damage normal testicular function.
- B.** Torsion can occur at any age; however, it is most common in adolescents, peaking at the age of 15 to 16 years.
- C.** Testicular torsion presents with sudden onset of pain and swelling in one testicle, occasionally associated with minor trauma. There is frequently nausea, vomiting, and lower abdominal or flank pain. There may be a history of previous similar episodes with spontaneous resolution.
- D.** A urinalysis is essential in differentiating testicular torsion from epididymitis; however, a negative urinalysis does not rule out epididymitis, since it may be of the sterile variant.

E. Differential Diagnosis of Testicular Torsion

- 1. Epididymitis due to *Neisseria gonorrhoeae* and *Chlamydia trachomatis* is much more common than torsion in adult men.
- 2. Torsion of an appendix testis or appendix epididymis may also mimic testicular torsion. A torsed appendix testis may manifest as a tender, pea-sized nodule at the upper pole of the testicle with a small blue-black dot seen through the scrotal skin (the blue dot sign); management is conservative. If there is diagnostic uncertainty, surgical exploration is required.
- 3. Other less common conditions that may present similarly to torsion include acute hemorrhage into a testicular neoplasm, orchitis, testicular abscess, incarcerated hernia, and testicular rupture.

F. Physical Examination

- 1. Testicular torsion usually presents with severe unilateral testicular pain with an acute onset. The pain is associated with an extremely tender testicle with a transverse lie or an anterior epididymis that lies high in the scrotum.
- 2. The testis is high in the scrotum (Bruns' sign). The presence of a cremasteric reflex almost always rules out testicular torsion.
- 3. Relief of pain by elevation of the affected testis (Prehn's sign) suggests epididymitis. A negative Prehn's sign (elevation of the testis does not relieve the pain) suggests testicular torsion.

G. Diagnostic Imaging Tests

- 1. Diagnostic testing should not delay surgical exploration in acute torsion. If the diagnosis is unclear, diagnostic tests may be useful.
- 2. Color Doppler ultrasound has emerged as the most valuable diagnostic study, with nearly 100% sensitivity and specificity.
- 3. Surgical exploration remains the best diagnostic tool.

H. Management of Testicular Torsion

- 1. Immediate detorsion is imperative for all cases of testicular torsion. Testicular salvage rates decrease to 50% at 10 hours and to 10-20% at 24 hours.
- 2. Manual detorsion can be attempted as an urgent measure by rotating the testicle about its pedicle. Surgical orchiopexy, however, is still required.
- 3. If an infarcted testicle is noted during surgical exploration, it should be removed. If the testicle is viable, both testicles should be fixed

in the scrotum with nonabsorbable sutures.

VI. Priapism

- A.** Priapism is defined as a prolonged penile erection.
- B.** Most cases of priapism in adults are idiopathic. However, priapism often occurs secondary to vasoactive injectable drugs used to treat impotence. In children the most common causes are sickle cell anemia, hematologic neoplasms (leukemia), and trauma.

C. Evaluation of Priapism

- 1. Patients usually complain of a persistent, painful erection. They may have fever and voiding difficulties.
- 2. Physical examination should include a neurologic evaluation and perineal inspection for neoplasms. Examination of the penis usually reveals a flaccid glans despite rigid corpora cavernosa.
- 3. Hematologic studies should be performed to rule out sickle cell anemia and leukemia.

D. Treatment of Priapism

- 1. Early treatment of priapism reduces the risk of fibrosis of the corpora cavernosa and subsequent long-term impotence, which may occur in up to 50% of patients.
- 2. Discomfort can be reduced with parenteral narcotic analgesics and sedation.
- 3. Detumescence may be achieved using cold compresses, ice packs, warm- or cold-water enemas, and prostate massage.
- 4. If these treatments are unsuccessful, the static blood may be aspirated from the corpora using a large bore needle. Followed by irrigation of the corpora with saline containing a vasoconstricting agent (phenylephrine, epinephrine, or metaraminol).
- 5. If this process fails to achieve detumescence, a shunt may be created between the affected corpora cavernosa and unaffected corpus spongiosum with a Tru-Cut biopsy needle.
- 6. When priapism is secondary to sickle cell anemia, therapy also includes hydration, oxygen, and blood transfusion.

Vascular Surgery

Ian L. Gordon, M.D., Ph.D.

Vertebrobasilar Insufficiency

I. Clinical Evaluation

- A.** Vertebrobasilar insufficiency is caused by atherosclerosis of the vertebral posterior circulation. Symptoms consist of intermittent dizziness, dysarthria, drop attacks, or vertigo caused by brainstem ischemia.
- B. Subclavian Steal Syndrome**
 - 1. Characteristic symptoms consist of dizziness, syncope, or visual disturbance with muscular exertion of the involved extremity. This syndrome is indicative of proximal subclavian artery occlusion.
- C.** Blood pressure difference >50 mmHg between arms may be noted.

II. Treatment of Vertebrobasilar Insufficiency

- A.** Vertebral artery reconstruction - Endarterectomy (removal of plaque) of the origin of the vertebral artery or transposition and reimplantation of the vertebral to the subclavian are options.

Peripheral Arterial Disease

Peripheral arterial disease is characterized by intermittent claudication, consisting of exercise-induced lower extremity pain that is relieved by rest. Claudication occurs when the blood supply is inadequate to meet the metabolic demand of lower limb muscles as a result of atherosclerotic arterial stenosis.

III. Pathophysiology

- A.** The incidence of claudication rises sharply between ages 50 and 75 years, particularly in persons with coronary artery disease.
- B.** This condition affects at least 10% of persons over 70 years of age and 2% of those 37 to 69 years of age.
- C.** Patients with peripheral arterial disease usually die of complications of coronary artery or cerebral arterial disease because of advanced atherosclerosis.
- D. Risk factors**
 - 1. Cigarette smoking is the most important risk factor. 70-90% of patients with arterial insufficiency are smokers. Risk remains increased for up to 5 years after smoking cessation.
 - 2. Other risk factors include hyperlipidemia, diabetes mellitus and hypertension.
- E.** Patients with claudication commonly manifest other symptoms of atherosclerosis, particularly coronary artery disease and cerebrovascular disease. The 10-year mortality rate from cardiovascular disease is 15 times higher in patients with claudication.

IV. Clinical Evaluation of Claudication

A. Initial evaluation consists of determining the location, extent, and severity of disease, and the degree of functional impairment.

B. Claudication

1. The key clinical features of claudication are the reproducibility of muscular pain in the thigh or calf after a given level of activity and cessation after a period of rest.
2. Patients should be asked about the intensity of claudication, its location, and the distance they have to walk before it begins.
3. **Aortoiliac disease** is manifest by discomfort in buttock and/or thigh and may result in impotence and reduced femoral pulses.. **Leriche's syndrome** occurs when impotence is associated with bilateral hip or thigh claudication.
4. **Iliofemoral occlusive disease** is characterized by thigh and calf claudication. Pulses are diminished from the groin to the foot.
5. **Femoropopliteal disease** usually causes calf pain. Thigh claudication will occur if the disease involves the common femoral artery and/or the profunda femoris artery. Patients have normal groin pulses but diminished pulses distally.
6. **Tibial vessel occlusive disease** may lead to foot claudication, rest pain, non-healing wounds, and gangrene.
 - a. **Rest Pain** consists of severe pain in the distal portion of foot due to ischemic neuritis. The pain is deep and unremitting, and it is exacerbated by elevation and relieved by dangling the affected foot over the side of the bed.

V. Physical Examination

A. Evaluation of the Peripheral Pulses includes the femoral, popliteal, posterior tibial, and dorsalis pedis arteries.

1. The quality of the pulse and any pulse asymmetries between the limbs should be noted. Absence of posterior tibial pulses is an excellent predictor of atherosclerotic disease.

B. Other signs of chronic arterial insufficiency include brittle nails, scaling skin, hair loss on the foot and lower leg, cold feet, cyanosis, and muscle atrophy. Careful inspection of the foot for skin breakdown or ulceration is important.

C. Bruits may be auscultated distal to the arterial obstruction.

D. The degree of collateral circulation may be estimated by elevating the leg until the foot becomes white, then lowering the leg and measuring the time required for capillary refill to occur (normal <5 seconds). More than 40 seconds indicates severe occlusive disease.

E. Abdominal examination for a "pulsatile mass" is performed because of the strong association between abdominal aortic aneurysm and peripheral arterial disease.

VI. Differential Diagnosis

A. Neurogenic Claudication (spinal stenosis)

1. Neurogenic claudication is the clinical syndrome most difficult to distinguish from claudication. Neurogenic claudication is caused by osteophytic narrowing of the neural canal around the spinal nerves.
2. This syndrome causes radicular leg pain that begins with a change of posture and is relieved by assuming the recumbent position. The

leg pain is often accompanied by burning, tingling, and numbness.

3. The pain of neurogenic claudication persists even after the patient has stopped walking or occurs with mere standing or after prolonged sitting. With neurogenic claudication, the distance walked until the onset of pain varies.
- B. **Nocturnal muscle cramps** occur in older persons, but the cramps are not related to exercise.
- C. **Chronic compartment syndrome** may occur in young athletes characterized by tightness and pain in the calf after vigorous exercise. The pain does not quickly subside with rest.
- D. **Osteoarthritis of the hip** may mimic thigh and buttock claudication. However, osteoarthritic pain occurs with variable amounts of exercise,, and the pain changes in severity from day to day.

VII. Laboratory Testing for Arterial Disease

A. Ankle-brachial Index (ABI)

1. The ankle-brachial index (ABI) is the ratio of ankle systolic blood pressure to arm systolic blood pressure, and it is highly sensitive and specific for peripheral arterial disease in nondiabetic patients.
 - a. 0.75 is considered normal
 - b. 0.40-0.75 suggests arterial obstruction with claudication
 - c. <0.40 suggests significant arterial obstruction with critical ischemia (i.e., non-healing wounds or rest pain).
2. Patients occasionally have normal ABIs at rest, but their symptoms strongly suggest claudication. In these patients, ABIs should be obtained before and after exercise. Exercise may unmask obstruction.
3. ABI and segmental blood pressure measurements are not accurate in patients with calcified vessels (especially diabetics) because ankle blood pressure readings in such patients tend to be falsely elevated.

B. **Color Doppler imaging** is a good diagnostic alternative for patients at high risk for calcification of vessels; it provides data on velocity as well as direction of blood flow; it has a sensitivity of 60-82% and a specificity of 92-98% for stenoses greater than 50%.

C. **Arteriography** is indicated following evaluation of ABIs if a patient with claudication is to undergo surgical or endovascular treatment.

D. **Magnetic resonance angiography** has superior sensitivity over conventional angiography in detecting disease in distal runoff vessels. The procedure is noninvasive.

VIII. Prognosis

- A. After five-years, 4% of patients with claudication lose a limb and 16% have worsening claudication or limb-threatening ischemia. Factors associated with the worsening of symptoms include cigarette smoking, diabetes, and severe arterial occlusive disease.
- B. The five-year mortality rate for patients with claudication is 29%; 60% of deaths result from coronary artery disease, 15% from cerebrovascular disease, and the remainder result from nonatherosclerotic causes.

IX. Nonoperative Management

A. Risk Factor Modification

1. **Cigarette smoking** is the most important modifiable risk factor. Claudication patients who abstain from tobacco usually do not progress to limb loss, whereas 11.3% of the patients who continue smoking require amputation.
2. **Meticulous care of skin** of the lower limbs helps avoid ulcer formation and skin infection.
3. **Obese patients** should be advised to lose weight through drugs, exercise, and dietary counseling.
4. **Hypertension** should be vigorously treated. Beta-blockers do not usually worsen claudication.
5. **Hyperlipidemia:** Regression of femoral atherosclerosis is possible with aggressive lipid-lowering therapy. The goal should be to lower the low-density lipoprotein cholesterol level to below 100 mg/dL.
6. **Diabetes Control**
 - a. Diabetes should be aggressively controlled in order to prevent further complications of microvascular disease.
 - b. Patients with diabetes often have peripheral neuropathy, and they require meticulous foot care to prevent diabetic foot ulcers.

B. Coronary and carotid artery disease assessment is essential because coronary disease accounts for 30% of deaths in patients with peripheral arterial disease.

C. Hormone replacement therapy: Postmenopausal hormone replacement may slow the progression of atherosclerosis.

D. Progressive Exercise Training

1. Patients who have intermittent claudication but no rest pain or ischemic ulceration should begin a walking program. They can usually increase their walking distance several times above baseline and can usually reduce the pain of claudication.
2. Patients should walk to or through the onset of claudication, rest until the pain resolves, and then resume exercise; 30 minutes to one hour every day is recommended.

E. Pharmacologic Management

1. Pentoxifylline (Trental)

- a. The effect of pentoxifylline is only modest. Patients who take it may have a 20% increase in walking distance; however, exercise alone may increase walking distance up to 25%. This drug is useful only in patients who cannot benefit from exercise.
- b. Pentoxifylline is given in a dose of 400 mg tid. Due to gastrointestinal side effects are common.

2. **Aspirin** has no effect on claudication, but because of the high incidence of cerebrovascular and cardiovascular disease, aspirin, 160-325 mg per day, should be given.

F. Operative and Endovascular Procedures

1. Most patients with claudication respond to conservative therapy. Surgery is reserved for patients with rest pain or tissue loss. Patients who have intermittent calf claudication alone are not surgical candidates unless the claudication severely limits their lifestyle or occupational functioning.
2. Patients with rest pain, tissue loss as a result of gangrene, or non-healing ulcers, with an ABI in the range of 0.3, are surgical candi-

dates and require formal noninvasive testing and arteriography.

3. Percutaneous transluminal angioplasty has a greater than 90% success rate in the treatment of short-segment aortoiliac occlusive disease, and these results may be improved with the placement of an intra-arterial stent. However, percutaneous transluminal angioplasty has shown five-year patency rates of only 40-60%.
4. Surgical bypass therapy is an effective treatment for claudication; however, it is associated with 5-10% morbidity and mortality rates. Aortobifemoral grafting has a 90% 5-year patency rate. Aortoiliac, femoral-femoral crossover, and reversed and in-situ saphenous vein bypass grafting from the common femoral to the popliteal artery have 60-70% 5-year patency rates. A synthetic polytetrafluoroethylene graft (PTFE) is indicated for above knee femoral-popliteal bypass, and it has a 50% 5-year patency rate.

G. Axillofemoral Bypass: This procedure is useful for high risk, elderly patients who are unable to tolerate an aortic procedure.

X. Management of the Acutely Threatened Limb

- A. An acutely occluded artery can result in limb loss within hours. The patient will complain of sudden onset of severe unrelenting rest pain. Atrial fibrillation often may cause an acute embolus to occlude the artery.
- B. These patients emergency surgical evaluation and immediate heparinization.

Abdominal Aortic Aneurysm

I. Clinical Evaluation

- A. The incidence of aortic dilation >4 cm is 1-2% in men over 65 years.
- B. **History:** Patient feels pulsatile abdominal mass. Suspect rupture or leak if hypotension, aneurysm tenderness or history of severe back, flank or abdominal pain.
- C. **Physical Exam:** Lateral pulsation, palpate dome of aneurysm below left costal margin, otherwise suspect thoracoabdominal component; check distal pulses and look for associated popliteal or femoral aneurysms.
- D. **Testing:** CT for preoperative evaluation and most accurate size measurement; ultrasound for screening and follow-up. Diameter of 5 cm in good risk patient is often used as indication for surgical repair.

II. Treatment

- A. Surgical therapy is generally indicated when the diameter is 5 cm or greater.
- B. Aneurysm replacement with Dacron graft (3-5% mortality).
- C. Endovascular graft placement for selected patients.

Deep Venous Thrombosis

I. Pathophysiology

- A. If venous thrombi of the lower extremities are not treated, 50% may embolize to the lung. The source of symptomatic pulmonary emboli is usually thrombosis in the larger veins above the knee rather than thrombosis in the veins of the calf.
- B. Deep venous thrombosis can be found in at least 80% of pulmonary emboli.

II. Risk Factors for Deep Venous Thrombosis

- A. **Venous Stasis:** Prolonged immobilization, stroke, myocardial infarction, heart failure, obesity, varicose veins, anesthesia, age > 65 years old.
- B. **Endothelial Injury:** Surgery, trauma, central venous access catheters, pacemaker wires, previous thromboembolic event (especially if precipitating factors are not eliminated).
- C. **Hypercoagulable State:** Malignant disease, high estrogen level (pregnancy, oral contraceptives).
- D. **Hematologic Disorders:** Polycythemia, leukocytosis, thrombocytosis, antithrombin III deficiency, protein C deficiency, protein S deficiency, antiphospholipid syndrome, inflammatory bowel disease.

III. Signs and Symptoms of Deep Venous Thrombosis

- A. Signs and symptoms of DVT are absent in about 50%, and they range from subtle to obvious.
- B. May be asymptomatic or the patient may complain of pain, swelling, "heaviness," aching, or the sudden appearance of varicose veins. Recognized risk factors may be absent.
- C. DVT may be characterized by an edematous limb with a erythrocyanotic appearance, dilated superficial veins, elevated skin temperature, tenderness in the thigh or calf. Absence of clinical signs does not preclude the diagnosis.
- D. A swollen, tender leg with a palpable venous "cord" in the popliteal fossa strongly suggests popliteal DVT. The lack of a palpable cord does not rule out DVT. Marked discrepancy in limb circumference supports the diagnosis of DVT, but most patients do not have measurable swelling.
- E. The clinical diagnosis of DVT is correct only 50% of the time; therefore, diagnostic testing is mandatory.

IV. Diagnostic Testing

- A. **Impedance Plethysmography** depends on the detection of impaired venous emptying of the leg. High false negative rate, and over 30% of cases may be missed. Plethysmography must be used in conjunction with duplex scanning.
- B. **Duplex Scanning:**
 - 1. **Doppler Studies:** Uses Doppler shifts to detect blood flow in the veins.
 - 2. **Ultrasound Imaging:** Two-dimensional ultrasonography produces an image of the deep veins.
 - 3. When Doppler measurement of blood flow is added to ultrasound

imaging, the combination is referred to as a duplex scan.

- C. When results of impedance plethysmography and duplex scanning are positive, these techniques are adequately specific. Results that do not support the clinical impression should be investigated with venography.
- D. **Contrast Venography:**
 - 1. When impedance plethysmography and ultrasound techniques fail to demonstrate a thrombus, venography is the diagnostic "gold standard" for patients at high clinical risk. The test is negative if contrast medium is seen throughout the deep venous system.
 - 2. Venography can cause iatrogenic venous thrombosis in 4%, and allergic contrast reactions occur in 3% of patients.
- E. MRI is a relatively new approach to the detection of DVT. MRI may have an accuracy comparable to that of contrast venography, and it may soon replace contrast venography as the "gold standard" of venous imaging.

V. Treatment of Deep Vein Thrombosis

A. Heparin Anticoagulation:

- 1. Continuous intravenous heparin is the standard of therapy for deep venous thrombosis at any site.
- 2. Anticoagulant therapy should be initiated as soon as venous thrombosis is clinically recognized as likely while awaiting the completion of a diagnostic workup.
- 3. The amount of heparin needed for effective anticoagulation is higher than was once taught; a 5,000-U bolus and a drip of 1,000 U/hr usually does not produce a therapeutic aPTT within the first 48 hours.
- 4. **Initial Bolus:** 10,000 U of heparin by intravenous push
- 5. **Initial Maintenance Drip:**
 - Mix 20,000 U of heparin in 500 cc of D5W or normal saline (40 U/cc)
 - Start drip beginning at 32 cc/hr (1,280 U/hr)
- 6. Check aPTT 6 hr after giving initial bolus. **Therapeutic Range:** Activated PTT 1.5-2.0 times control.

Maintenance Dose Adjustment:

aPTT	Hold drip	Adjust drip	Check aPTT
<50 sec	0 min	Increase 3 cc/hr	6 hr later
50-59 sec	0 min	Increase 3 cc/hr	6 hr later
60-85 sec	0 min	No change	Next morning
86-95 sec	0 min	Decrease 2 cc/hr	Next morning
96-120 sec	30 min	Decrease 2 cc/hr	6 hr later
>120 sec	60 min	Decrease 4 cc/hr	6 hr later

- 7. Heparin must be continued until the anticoagulant effects of warfarin have become established (usually 3 to 5 days after initiation). Subcutaneous low-molecular weight heparin may replace intravenous heparin in the next few years but is not generally available at present.
- 8. Monitor platelet count during heparin therapy; thrombocytopenia develops in up to 5%. Heparin rarely induces hyperkalemia, which resolves spontaneously upon discontinuation. Ambulation can begin once the activated PTT is in the therapeutic range.

B. Oral Anticoagulants:

1. Oral anticoagulants should be started only after effective anticoagulation with heparin has been established because they increase coagulability and thrombogenesis during the first few days of administration. There is a high incidence of progressive thrombosis and embolization when oral warfarin is started without adequate prior heparinization.
2. **Warfarin (Coumadin)**, initial dose 10 mg; subsequent doses should be based on a daily PT values and its rate of change.
3. After heparin therapy has overlapped with warfarin therapy for 3-5 days and the PT is 1.3-1.5 times control (or International Normalized Ratio 2.0-3.0), heparin can be safely discontinued. Outpatients receiving oral anticoagulants should have their PT checked weekly.
4. **Length of Warfarin Therapy:** Oral anticoagulation should be maintained for 3 months after a first episode of DVT or pulmonary embolism. Indications to prolong therapy to 6-12 months include persistent risk factors, extensive or complicated thrombosis, or recurrent thrombotic episodes.

C. Thrombolytic Therapy:

1. Thrombolytics clear the thrombus more rapidly, and preserve venous valvular function.
2. **Indications for Thrombolytics in DVT:**
 - a. Patients with extensive DVT.
 - b. Those with severe venous outflow obstruction (phlegmasia cerulea dolens).
3. **Contraindications to Thrombolytics:**
 - a. **Absolute Contraindications:** Active bleeding, recent stroke, central nervous system tumor.
 - b. **Relative Contraindications:** Surgical procedure within preceding 10 days; recent gastrointestinal bleeding; uncontrolled hypertension; recent trauma (cardiopulmonary resuscitation), pregnancy, endocarditis, renal or hepatic failure, left-sided heart thrombus.
4. The thrombus should usually be demonstrated by venography before thrombolytics are used.
5. The agent should be delivered as close to the thrombus as possible. Catheter-induced thrombosis in the axillary or subclavian vein or superior vena cava can best be lysed by direct infusion through the involved catheter.
6. Monitor complete blood count, PT or international normalized ratio (INR), activated PTT, and fibrinogen levels twice daily.
7. **Streptokinase:** Duration required for complete clot lysis is substantially longer with streptokinase than with tissue plasminogen activator (tPA); much less expensive than tPA. **Dosage:** bolus of 250,000 U IV followed by 100,000 U/hr
8. **Tissue Plasminogen Activator:** tPA is "clot-selective", and may promote clot lysis more rapidly than other agents. **Dosage:** 100 mg over a 2-3 hour period.

D. Thrombectomy for DVT may be indicated for phlegmasia cerulea dolens (severe DVT) after thrombolytic therapy has failed.

E. Vena Caval Interruption Methods:

1. Pulmonary embolism can be prevented by percutaneous filter

interruption of the inferior vena cava. Filters have a patency rate of 95% and are effective in preventing 95% of emboli.

2. **Indications for Vena Cava Filter:** Pulmonary embolism despite adequate anticoagulation, contraindications to anticoagulants, prevention of recurrent pulmonary embolism in high-risk patients.

VI. Superficial Non-Septic Phlebitis

1. Patients with isolated thrombosis in a calf vein or with purely superficial phlebitis should remain ambulatory and wear compression stockings.
2. Patients with superficial non-septic phlebitis, in whom ultrasound imaging has excluded deep-system thrombosis, may be treated with nonsteroidal anti-inflammatory drugs.

VII. Prevention of Deep Vein Thrombosis

- A. Prophylaxis should be given to patients at risk (most hospitalized patients).
- B. Low dose of heparin (5,000 U subcutaneously every 12 hours) or pneumatic compression stockings are effective in reducing the occurrence of thrombosis.

Orthopedic Surgery

Harry Skinner, M.D.

Management of Orthopedic Fractures and Dislocations

I. Clinical Evaluation of the Injured Limb

A. Physical Examination of the Injured Limb: 1) inspection-look, 2) palpation -- feel, and 3) movement -- move. Specifically examine the bone for instability and examine the soft tissue for associated injury. Evaluate and document neurologic and vascular status.

B. Clinical Features of Fractures

1. **Pain and Tenderness:** All fractures cause pain in the neurologically intact limb.
2. **Loss of Function:** Pain and loss of structural integrity of the limb causes loss of function.
3. **Deformity:** Change in Length, Angulation, Rotation and Displacement.
4. **Attitude:** The position of the fractured limb is sometimes diagnostic. For example the patient with a fractured clavicle usually supports the limb and rotates his head to the affected side.
5. **Abnormal Mobility and Crepitus:** Eliciting these signs may be painful and are potentially dangerous, they should not be sought deliberately.

C. Clinical Features of Dislocations

1. A dislocation occurs when the articular surfaces of a joint are no longer in anatomic contact. Subluxation (partial dislocation) is a less severe condition that occurs when the orientation of the surfaces is altered but they remain in contact.
2. **Pain and Tenderness:** Severe pain may be completely relieved when the joint is relocated.
3. **Loss of Motion:** Both active and passive motion are grossly limited in all dislocations.
4. **Loss of Normal Joint Contour:** In the anteriorly dislocated shoulder, the deltoid is flattened and the greater tuberosity of the humerus is no longer lateral to the acromion.
5. **Attitude:** The patient carefully holds the anteriorly dislocated shoulder in abduction and external rotation.
6. **Neurologic Injury:** The incidence of neurologic injuries is much higher with dislocations than with fractures. Shoulder dislocations are often associated with axillary nerve injury. Posterior dislocations of the hip can result in sciatic nerve contusion. Careful examination for neurologic status is indicated before any intervention. If a neurologic injury is caused by the reduction maneuver, immediate open. exploration is indicated.

II. Clinical Description of Fractures

A. Anatomic Location

1. **Metaphyseal** -- Fracture through metaphysis
2. **Diaphyseal** -- Fracture through diaphysis (proximal, middle or distal third of the shaft)
3. **Epiphyseal** -- Fracture through epiphysis or physis
4. **Salter Classification of Fractures (children only):**

Type I: Fracture through physis, between the epiphysis and metaphysis

Type II: Fracture through physis, involving the metaphysis

Type III: Fracture through physis, involving the epiphysis

Type IV: Fracture through physis, involving the metaphysis and epiphysis

Type V: Crush injury to physis, between the epiphysis and metaphysis

B. Bony Deformity: Describe any change in bone Length, Angulation, Rotation, and Displacement (LARD).

C. Direction of the fracture line: Describe the radiographic direction that the fracture travels.

1. **Transverse** -- Perpendicular to the long axis of the bone
2. **Oblique** -- Plane of fracture is at some angle to the bone
3. **Spiral** -- Secondary to torsional stress

D. Comminution: Fracture with more than two fragments.

E. Open vs Closed: In an open fracture the bone protrudes through the skin. The terms compound and simple are no longer used.

F. Special Fracture Types

1. **Pathological Fracture:** The bone is weakened by preexisting disease such as osteoporosis or a tumor.
2. **Stress Fracture:** Bone can become fatigued by repetitive stress, and a fracture may develop.
3. **Buckle Fracture:** One cortex is broken while the other remains intact.

III. Radiologic Evaluation of Fractures

A. A minimum of two views at right angles to each other should be obtained. Visualize the joint above and below the injury and check for soft tissue swelling.

B. Views of the uninjured extremity are often useful for comparison in children.

IV. Management of Fractures

A. Fracture Reduction

1. The fracture must be restored to a normal anatomical position without allowing bones to override.
2. Muscle spasm should be relieved with traction, analgesics, muscle relaxants.
3. Bones must be in apposition; properly align bone in linear and rotary directions and set to proper length.

B. Indications for Operative Treatment

1. **Failure of closed methods** to reduce the fracture.
2. **Multiple Injuries:** Multiple fractures should be fixed internally.
3. **Early Mobilization:** Associated medical problems and high cost

can be avoided by early mobilization.

4. **Fractured and Displaced Articular Surfaces:** Operative treatment minimizes the risk of late degenerative changes.
5. **Fractures Secondary to Tumor Metastasis:** Internal fixation allows the patient to return home more rapidly.
6. **Associated Arterial Injury:** Internal fixation stabilizes the limb after arterial repair.

C. Upper Extremity Fractures and Dislocations

1. **Clavicle Fractures:** Cannot be casted. Subclavian artery and brachial plexus injury may occur. Splint with figure-of-eight bandage, or if figure of eight causes posterior angulation, use triangular sling.
2. **Humerus Fractures:** X-ray the entire bone, and check for radial nerve injury (wrist drop). Treat with collar-and-cuff sling; immobilize the joints above and below fracture.
3. **Anterior Shoulder Dislocation:** The humeral head has been forced anterior to glenoid. Usually caused by extension force applied to abducted arm. Patient presents with arm in slight abduction and cannot bring elbow to side; there is a slightly depressed deltoid prominence, and arm motion causes pain. Axillary nerve injury may occur, causing a sensory deficit over the deltoid. Reduce with gentle traction.
4. **Posterior Shoulder Dislocation:** The humeral head has been forced posterior to the glenoid. May be secondary to seizures or electrocution.
5. **Colles Fracture:** Distal radius is angulated dorsally; associated fracture of the ulnar styloid. Treatment consists of reduction, casting, and elevation of the extremity until swelling subsides; check alignment with a postreduction x-ray; rule out median nerve injury by examination.
6. **Smith Fracture:** Volar angulation of fractured radius (reverse Colles). This fracture should be reduced and cast in the same manner as a Colles fracture in slight flexion. Frequently surgical fixation is necessary because the fracture is unstable.
7. **Scaphoid (navicular) Fracture:** Often occurs after fall on outstretched hand; tenderness occurs over the anatomic snuff-box. X-rays frequently are negative initially. If scaphoid fracture is clinically suspected, immobilize wrist and x-ray in 2-3 weeks.

D. Lower Extremity Fractures and Dislocations

1. **Femoral Neck Fractures:** Osteoporotic bone predisposes to this intracapsular fracture. Internal fixation or endoprostheses (artificial hip) are required.
2. **Intertrochanteric Fractures:** Usually occurs in elderly after a fall out of bed. The fracture is located on the outside of the joint capsule. Internal fixation is required.
3. **Femoral Shaft Fracture:** May occasionally cause an arterial injury. Early intermedullary nailing is recommended in adults.

E. Knee Ligament Testing

1. **Varus/Valgus Stress of the Tibia on the Femur:** Examiner stabilizes the femur, and pressure is exerted outward or inward at the ankle; a tear of the collateral ligament is indicated by excess mobility.
2. **Anterior Drawer Test:** Pull tibia anteriorly with the knee flexed 90° (tests for tear of anterior cruciate).

3. Posterior Drawer Test: Push tibia posteriorly with the knee flexed 90° (tests for tear of posterior cruciate).

4. Knee Injuries

a. The most common ligamentous injuries are tearing of the medial collateral ligament by a blow from the lateral side of the knee, or tear of the anterior cruciate ligament injury by twisting on a planted foot. Brace immobilization is usually sufficient, but recurrent instability requires surgical repair.

b. Dislocation of the knee often results in multiple ligament injury. Rule out popliteal artery trauma. Immobilization of the knee, followed by ligament reconstruction should be completed.

F. Ankle Fractures

1. Lateral displacement of the talus from its position below the tibia is associated with a displaced fracture of the lateral malleolus.

2. Reduction requires anesthesia and internal fixation of the talus; check anteroposterior lateral, oblique, and stress x-rays views to confirm realignment.

Degenerative Joint Disease

Osteoarthritis (osteoarthrosis) refers to a group of noninflammatory arthritides which are the end of a common pathway resulting from injury to articular cartilage. The term is synonymous with degenerative joint disease (DJD). Generally the prevalence of DJD increases with age, and the most common associated factor is trauma.

I. Treatment of Degenerative Joint Disease

A. Modification of activities, weight reduction and physical therapy should be advised. NSAIDs are valuable in reducing symptoms.

B. Patients respond differently to different NSAIDs, so one drug may be more efficacious than other in a particular patient. Peptic ulcers develop in approximately 1% of patients taking NSAIDs.

C. For small joints of the hand and foot, fusion or resection of the diseased joint generally permits pain-free function, and should be done when symptoms become disabling. For the large weight bearing joints of the lower extremity (hip and knee), fusion is generally done only when joint replacement has failed.

D. Tibial osteotomy is the treatment of choice for DJD of the medial tibial plateau of the knee. Tibial osteotomy is performed in the young (<60) patient with varus knees and isolated medial joint arthritis. Results are best when the procedure is performed early in the course of the disease. The procedure is accomplished by removing a wedge of bone from just under the plateau, such that a varus knee becomes a valgus knee, shifting the weight to the lateral side.

E. Total joint arthroplasty (TJA) continues to provide excellent results in over 90% of patients at 10 years. Joint replacement (total hip, total knee) is performed for incapacitating pain and loss of ability to walk 2 blocks.

F. Treatment of DJD of the low back is initially non-operative (weight reduction, NSAID's, physical therapy, orthoses). Incapacitating back pain from facet arthritis and disc degeneration can justify arthrodesis

with the placement of metal instrumentation to improve fusion rates.

$$\begin{aligned} A-a \text{ gradient} &= [(P_B - PH_2O) FiO_2 - PCO_2/R] - PO_2 \text{ arterial} \\ &= (713 \times FiO_2 - pCO_2/0.8) - pO_2 \text{ arterial} \end{aligned}$$

$P_B = 760 \text{ mmHg}$; $PH_2O = 47 \text{ mmHg}$; $R \approx 0.8$
normal Aa gradient $<10\text{-}15 \text{ mmHg}$ (room air)

Arterial oxygen capacity $= (\text{Hgb}(\text{gm})/100 \text{ mL}) \times 1.36 \text{ mL O}_2/\text{gm Hgb}$

Arterial O₂ content $= 1.36(\text{Hgb})(\text{SaO}_2) + 0.003(\text{PaO}_2) = \text{NL } 20 \text{ vol\%}$

O₂ delivery $= \text{CO} \times \text{arterial O}_2 \text{ content} = \text{NL } 640\text{-}1000 \text{ mL O}_2/\text{min}$

Cardiac output $= \text{HR} \times \text{stroke volume}$

$$\begin{aligned} \text{CO L/min} &= \frac{125 \text{ mL O}_2/\text{min}/\text{M}^2}{8.5 \{ (1.36)(\text{Hgb})(\text{SaO}_2) - (1.36)(\text{Hgb})(\text{SvO}_2) \}} \times 100 \\ &(\text{need to multiply by M}^2 \text{ to get CO estimate for a patient. This is CI estimate}) \end{aligned}$$

Note: 125 is a crude estimate for normals
Normal CO $= 4\text{-}6 \text{ L/min}$

$$\text{SVR} = \frac{\text{MAP} - \text{CVP} \times 80}{\text{CO L/min}} = \text{NL } 800\text{-}1200 \text{ dyne/sec/cm}^2$$

$$\text{PVR} = \frac{\text{PA} - \text{PCWP} \times 80}{\text{CO L/min}} = \text{NL } 45\text{-}120 \text{ dyne/sec/cm}^2$$

$$\text{Creatinine clearance} = \frac{\text{U Cr (mg/100 mL)} \times \text{U vol (mL)}}{\text{P Cr (mg/100 mL)} \times \text{time (1440 min for 24h)}}$$

Normal creatinine clearance $= 100\text{-}125 \text{ ml/min (males), } 85\text{-}105 \text{ (females)}$

Anion Gap $= \text{Na} + \text{K} - (\text{Cl} + \text{HCO}_3)$

Corrected serum Ca^+ (mg/dl) $= \text{measured Ca mg/dl} + 0.8 \times (4 - \text{albumin g/dl})$

Basal energy expenditure (BEE):

Males $= 66 + (13.7 \times \text{actual weight Kg}) + (5 \times \text{height cm}) - (6.8 \times \text{age})$

Females $= 655 + (9.6 \times \text{actual weight Kg}) + (1.7 \times \text{height cm}) - (4.7 \times \text{age})$

Nitrogen Balance $= \text{Gm protein intake}/6.25 - \text{urine urea nitrogen} - (3\text{-}4 \text{ gm/d insensible loss})$

Shock Characteristics:

	<u>CVP</u>	<u>Wedge</u>	<u>SVR</u>	<u>Cardiac Output</u>
LV Failure	NL or ↑	↑	↑	↓
Hypovolemia	↓	↓	↑	↓ or NL
Septis	↓	Variable	↓	↑

Cardiac Index (CI) Normal $= 2.5 \text{ to } 3.5 \text{ L/min m}^2$

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